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AN OUTLINE

OF

INFLAMMATION AND ITS EFFECTS:

BEING

FIRST PRINCIPLES OF SURGERY.

BY

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TO

STUDENTS OF SURGERY,

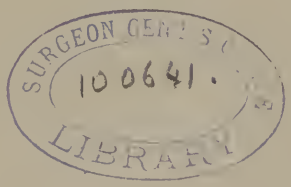
THIS WORK,

INTENDED TO ASSIST THEM IN THEIR LABOURS,

IS RESPECTFULLY DEDICATED

BY

THE AUTHOR.



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INTRODUCTION.

The first principles of Surgery with which the student should make himself familiar are those relating to inflammation and its effects. An intimate acquaintance with this subject is of as great importance to the practical surgeon as that of fever to the practical physician. Independent of any interest attached to the investigation of its phenomena, the researches of modern pathologists have shown that disordered vascular action forms the ground-work of many diseases, and, at some period or other, accompanies almost all. Diseases may be of themselves purely inflammatory; or inflammation may be united with them, or supervene during any part of their course. Many cases which formerly went under the very convenient title of *nervous*, as, for example, *amaurosis*, are now known to be in general the result of inflammation or its effects; and hence, an opposite and more successful line of treatment has been adopted. So readily is this species of morbid action kindled up, and so widely diffused is the structure in which it has its seat—wherever, indeed, vascularity can be detected—that we need not wonder at its so frequent occurrence. The time is probably not far distant when the nature of disease will be better understood; and when inflammation will be allowed to hold a more prominent share in its constitution than is even yet assigned to it.

By the surgeon, inflammation may be considered under two points of view—either as a morbid process, requiring its approach to be carefully watched and every means adopted to keep it in subjection; or, as an act of restoration on the part of nature to repair injuries or get rid of foreign bodies introduced within the system. This apparent discrepancy, with respect to the ultimate effects produced by inflammation, gave rise to a discussion amongst some of the older writers, as to whether it should be viewed as a salutary or destructive process. The question may be readily and satisfactorily answered. When inflammation takes place in any of the great cavities of the body, from the application of cold to the surface,

it may, if unchecked, either lead to the loss of life or so impair the structure of organs as to render them ever afterwards incapable of performing their proper functions; hence it is assuredly a morbid action intended to answer no good end, and causes the most serious and fatal consequences. On the other hand, how highly useful, nay, indispensably necessary, is inflammation? Without it, the progress of mortification could never be arrested and the dead parts cast off from the living; the mouth of a bleeding artery could not be permanently closed; no collection of purulent matter or foreign body travel to the nearest outlet and be discharged; no chasm made by ulceration filled up; no breach of surface repaired; no wound healed; and hence, no operation attempted. We may go a step farther, and state that, besides the salutary effects thus naturally obtained, inflammation is frequently induced by the surgeon where he wishes to obliterate some cavity or canal, as in the radical cure of *hydrocele* or in the treatment of a *sinuous* ulcer. Suited for the purposes now mentioned, inflammation must always be of a limited and peculiar character; when too sluggish, it requires to be urged on; when too violent, it must be repressed. We are, therefore, taught that inflammation is the most fatal of all diseases; and, in fact, with a few exceptions, all diseases threatening speedy destruction to life spring from it; while, again, it is the only way by which lost parts can be restored, or the effects of injuries repaired. Without it, in short, the business of the surgeon would, in many cases, be at an end.

In calling the student's attention to this subject, we have never deemed it necessary to make any apology for the length of time occupied in its discussion, as we consider that on this knowledge depends, in a great measure, the success of his future labours. We have no hesitation in stating, from our own individual experience, that one of the greatest errors committed in the first acquirement of a surgical education is the hurrying over doctrines which appear dry and uninteresting, and passing on to the consideration of injuries and operations, without stopping to contemplate what it is that renders the former so dangerous or leads so often to the necessity for the latter. In this way, the separate parts of surgery are eagerly sought after, while its fundamental principles are neglected; to use a metaphor, the branches are cultivated while the parent trunk, around which they entwine, lies forgotten. But let the student turn in what direction he pleases, inflammation continually crosses his path; it presents itself wherever he goes; its effects are revealed to him in every new state which comes under his notice; it is to

prevent its inroad and subdue it when it has appeared, that he observes the experienced surgeon manifesting such anxiety; and it is the great agent against which he is admonished he has to contend in the ordinary routine of daily practice. We cannot afford space to enter into minute particulars; but let him select any subject which is considered to belong to this branch of his profession; let him turn to wounds and their consequences; diseases or injuries of the coats of the vessels; those of the bones or joints; of the urinary organs; the organs of sense; the head, chest, or abdomen; or any of the various operations required, and we think a little reflection will teach him the intimate connection between all these and inflammation—the necessity, therefore, for the minute and careful study of the latter—and the impossibility of making any permanent and valuable progress without such fundamental instruction. Wherever, indeed, we have found this rule reversed, there crude and ignorant notions were sure to prevail.

Were we to descend from what experience has taught us and be required to bring forward authority in vindication of the position we have taken up, we would gladly appeal to one of the greatest names left us on earth; nor, under the present circumstances, could we stimulate the student to industry, should we be stepping far out of our path in bringing before him a few of the most remarkable features in the history of John Hunter. Here he is furnished with an instance of an individual, born in an obscure parish, receiving scarcely the rude outlines of a common education, and bred at first as a carpenter, who, by self-tuition, perseverance, and unwearied application, gradually rose in life till he assumed the most conspicuous place in the metropolis of his country. We might, therefore, dwell on his professional industry to illustrate what ours should be, or hold up his limited means and humble origin as an example for all to persevere. To his unceasing labours, Hunter was mainly indebted for the distinction he acquired; and, with the exception of a few hours allowed for repose, his indefatigable mind seems to have been ever at work. He has done, in many respects, for our profession, what Newton did for natural science. He was constantly bent on its improvement; and, in every department to which he turned his attention, we discover the traces of his masterly hand. The great monument of his industry, and that which will hand down his fame to all future ages, has fortunately been preserved; nor was it a mind of any ordinary description which thus ventured to shadow forth in one vast series the universe of life. The conception, it has been said, was worthy of Milton; the labour

could have been achieved by few men except Hunter. Volumes have been written on his genius; orations have been spoken in his praise; but one glance round the gallery of his Museum is worth them all. Where we asked for Hunter's epitaph, we would lead the enquirer into this noble edifice and say—

"Si monumentum quæras, circumspice."

Such men as Hunter appear only once during the existence of a world; and it is no easy task for posterity to speak concerning them as they deserve. The former reflection is startling but correct. Thus, while Newton, Locke, Bacon, Shakspeare, Scott, and many others, are infinitely removed from the rest of mankind, Hunter stands alone in our profession. When we think of all that he has accomplished, we are almost tempted to regard him as supernatural. His life resembles the transit of a comet, which bewilders while it excites admiration; which dazzles with excess of brilliancy, and then for ever disappears. How humble do any of the men of the present day appear when placed by the side of Hunter. In his immense career, every thing bore reference to one great idea—the discovery and elucidation of nature's laws. From the course of investigation which he pursued, he stepped at once out of the night of darkness into the full sunshine of knowledge. The difficulties he had to contend with, only add an intenser lustre to the glory he has acquired; and any imperfections we now perceive in his writings are as the shadows of a great landscape, rendering its prominent objects more distinct. By his own exertions alone, he liberated many parts of surgery from the impurities by which they were disfigured. On the lofty elevation reared by his own labours, he stood pre-eminent; and his history records all that gives life a value, and refines and ennobles the species. The peculiar character which distinguishes any past generation must always be interesting to that which follows, and may afford matter for much useful discourse; but the peculiar character of man, and of the mind of man—for ever active, yet for ever varying—is a theme of more permanent utility and sublimer interest. Here our brief sketch must close. Earthly fame is too often like a withered leaf, at one time tossed by the winds, at another, trodden under foot of men; but the grave of Hunter will be for ever clad with the verdure of immortality.

The importance which Mr. Hunter attached to the study of inflammation may be gathered from the eagerness with which he pursued the investigation of its phenomena. What skill he possessed in surgery is mainly to be attributed to his accurate know-

ledge of anatomy, and the profound views he entertained of disease. His treatise on "Blood and Inflammation" is invaluable, and will always be a lasting mark of his genius and reputation. In it he brought to light much that was unknown or misunderstood, and he stripped other parts of the absurd explanations with which they were encumbered. As in all his other works, we find him taking the deepest and most original views of morbid action, founded on the closest observation and experiment; for, without these, Mr. Hunter was, fortunately, not much addicted to theory. The most interesting chapter is that on "The Adhesive Inflammation," and which contains all that is known on this subject in the present day. Next we might reckon that on ulceration and the different kinds of absorption which go on in the healthy and morbid states of the body; but it is needless to make selections from a work replete with interest, and which should be carefully and frequently read by every candidate for surgical fame.

Since Mr. Hunter's time, many valuable observations have been made on inflammation, especially on the nature of the morbid action itself; nor, even now, does it appear a little singular that this great man should have contented himself with reasoning on the state of the vessels rather than attempting to ascertain by experiment their actual condition. On this head, we are deeply indebted to the early investigations of Drs. Philip, Thomson, and Hastings, and, more recently, to the philosophical and accurate researches of Gendrin and Kaltenbrunner. Nevertheless, much difference of opinion still exists, and much therefore remains to be accomplished. The more immediate phenomena of inflammation are so intimately connected with the structure and functions of the minute vessels, that it is impossible to understand the one without a constant reference to the other; and before the student looks narrowly into this part of the subject, we would recommend him to have his mind rightly informed respecting the capillary circulation. Were the physical and physiological properties of the arterial system better known, we should have much less difficulty in answering the question, "what is the condition of the vessels during inflammation?" and to this want of knowledge do we attribute all the errors which have been committed, and all the useless discussions which have arisen. The investigation might, in truth be so far defined, as an examination into the difference between the healthy and morbid states of the capillary branches; and hence the necessity of having a just conception of the one before we pretend to judge of the other. In order that we might not be misunderstood on this intricate

question, we have taken the liberty of making a few preliminary remarks on the nature of the vessels by which the first part of the circulation is carried on. In the brief outline there given, we have only alluded to those points which are essentially connected with the phenomena of inflammation; and, we may farther add, that the statements contained in that and the succeeding section are derived either from our own or the experiments of others.

In the following pages, it has been our aim to render a simple statement of known or acknowledged facts: they may, therefore, contain nothing new or very striking. We have likewise, as far as possible, endeavoured to avoid controversy, except where such was absolutely demanded; and then we have generally assigned what appeared to us the strongest reasons for believing in the particular view set forth. To prevent unnecessary reference to authorities, it will be seen that we have consulted the writings of Mr. Hunter and Burns, Drs. Philip, Thomson and Hastings, and Gendrin and Kaltenbrunner. There are others of minor importance to which we may also have occasionally referred. In conclusion, our object in publishing this work has been, first, to enable us to abridge the early part of our lectures, and allow longer time for the discussion of other important subjects; and, secondly to furnish the surgical student with an outline of inflammation and its effects brought down to the present period.

FIRST PRINCIPLES OF SURGERY.

SECTION I.

OF THE ARTERIAL SYSTEM.

The arterial system is made up of a series of cylindrical tubes, through which the blood is transmitted to all parts of the body. Each artery divides into two or more smaller branches, which again subdivide into others; so that the vessels, as they recede from the heart, diminish in size but increase in number. The arteries thus constitute so many descending cylinders, having an arborescent or conical form, with the apex towards the heart. The sum of the areas of the branches into which any vessel divides is always greater than the trunk from which the branches proceed. On this subject, no accurate calculation has been made; but, according to Haller, it may be stated nearly as one to one and a half. The ultimate terminations of arteries are so minute as to escape detection with the naked eye. They have, accordingly, received the name of capillary—a phrase in daily use, yet one to which no accurate definition has been affixed. The same general observations apply to the distribution of the veins, although these are more numerous and larger than the arteries. In estimating the size of the veins, we, of course, begin by tracing them from their origin in invisible vessels, to their union or termination in the larger trunks; hence we state, that the sum of the areas of the small veins exceeds that of the superior or inferior cava. These points are of great importance to remember, when attempting to estimate the velocity of the blood's motion in the different series of vessels; as, according to the ordinary laws of fluids, it must be slower in the capillary arteries and veins than in the larger trunks of either system.

When an artery of the middle size, as the popliteal, is examined, it is found to consist of three distinct tunics, to each of which considerable interest is attached, because on them depend the physical and physiological properties of the vessel. The first or outermost coat, named also the cellular or elastic, is made up of dense white fibres, closely compacted together. It is susceptible of great distension, as evinced in cases of aneurism—highly elastic, especially in the longitudinal direction, as proved by cutting an artery across;

and of such firm structure, that a ligature drawn tightly around fails to divide it. It varies much in thickness in different arteries, and even in different branches of the same artery; circumstances which have not been sufficiently acknowledged. Thus it is more abundant in the aorta and large vessels, where elasticity is required; while it seems wanting in the arteries of the brain. It is connected to the common sheath which envelopes arteries by means of fine cellular tissue, which is of great utility in checking hemorrhage when the vessels are cut across; and into the cells of which is secreted a serous fluid in the young, and an oily-looking substance in the adult subject.

On dissecting off the outer tunic, we arrive at a number of fibres, placed obliquely round the artery, constituting segments of circles so united as to form rings, having a yellowish hue in the larger, and a reddish colour in the smaller vessels, and divisible, in the former, into several layers. These constitute the middle or fibrous tunic; or, as it has been named by some, the muscular or proper membrane of arteries. It is distinguished from the outermost coat by its fibrous appearance, by the difference in its distribution throughout the vessels, and by its physical and organic properties. The fibres are flattened, dense, elastic, and easily divided by the pressure of a ligature. They are much more apparent in the small than in the large vessels; and, therefore, proportionally thicker in the former than in the latter, being opposed in this respect to the external tunic; they cannot, however, be traced in arteries less than a line in diameter, and seem to be also wanting in those of the brain. The identity of the fibres of the middle tunic with those of muscle, has, for many years, been a subject of keen dispute. After a repetition of some of the experiments, and a careful consideration of the arguments on both sides, we have been forced to the conclusion, that the arterial fibre has nothing in common with that of muscle. We will only allude, at present, to a few of their physical differences. In the first place, it has been sufficiently proved that the structure of artery contains no fibrin, a substance which enters so abundantly into the formation of muscle. Beclard, indeed, has stated, in his treatise on general anatomy, that he discovered fibrin in the composition of artery; but he hesitates to consider it as proper muscular or fibrinous tissue. Berzelius and Dr. Young have denied the existence of fibrin in the arterial tunics; and we are not aware that the result of their analysis has been contradicted by any succeeding chemist. Other striking proofs of the difference in the composition of artery and muscle are afforded by the application of alcohol, diluted acids, or hot fluids, not corrosive in their nature. All these agents shrivel and harden the arterial tissue—a phenomenon depending on the more complete coagulation of one of its chemical principles (albumen); while they exert no change on the muscular substance. The fibres of muscle are also much looser in structure than those of artery; neither do they possess the elasticity so conspicuously

seen in the latter. The effects of compression are widely different. When muscular fibres are surrounded with a tight ligature, they undergo a sort of strangulation; when the experiment is repeated on those of arteries, they are divided or cut. For these and other reasons, we dissent from the doctrine of the arterial and muscular fibres being one and the same; a doctrine which has been too readily, and without due precaution, admitted.

The third or internal coat of arteries is their lining membrane, thin, smooth, and transparent; and unlike either of the others, by extending uniformly throughout the whole system. It is readily divided by the ligature, neither admitting of extension, or possessing elasticity; and, although apparently weak, is yet strong enough to resist dilatation from the impetus of the blood after the outer tunics have been dissected off. In properties, it resembles the serous membranes; being moistened by a watery fluid in health, and throwing out adhesive lymph when attacked by inflammation. The arterial system thus made up, is admirably adapted for conveying the blood to the most distant parts of the body. The strength of the different branches may be conceived from the experiment of Dr. Hales, in which a force equal to a column of water one hundred and ninety feet high was required for bursting the carotid of a dog. Under this pressure, the vessel did not previously dilate, but burst at once; and from which it may be inferred, that dilatation of the coats of arteries, as in aneurism, can only be effected gradually and by length of time.

The arterial tissue is highly organised, being supplied with blood-vessels, nerves, and absorbents The nutritious arteries, the *vasa vasorum*, as they are named, are remarkable for coming from a neighbouring branch, and not from the trunk itself which they supply; they anastomose very sparingly with those either above or below, and can be traced only to the fibrous tunic, although the effects of disease show the lining membrane to be also copiously supplied. The nerves are principally derived from the ganglions of the great sympathetic, and partly also from the cerebro-spinal system; they can only be distinctly traced on the larger branches, and seem to be lost in the second tunic. It is important to recollect the origin and distribution of the nerves, as we will have occasion to refer to them hereafter. Lymphatics cannot be demonstrated either by injection or the microscope; but their presence is proved from the ulcerations seen in the inner coats of arteries, and from the manner in which a ligature is detached.

Before speaking of the physiological properties of the arteries, let us briefly allude to what is known respecting their terminations. When an artery, according to Haller, is traced to its final division, it will be found to end—either in a red vein or veins—in an excreting duct, as in any of the glandular organs—in an exhalant, as on the skin or a serous membrane—in very minute vessels, as lymphatics—or in a colourless vessel, *arteria non rubra*, through which red blood does not circulate in the healthy state. The

ultimate distribution of arteries is discovered by injecting the vessels with quicksilver in the dead body, or by examining them with the microscope in the living; and both of these inform us that arteries terminate in red veins. But although either method affords the only proof on which we can depend, certain changes going forward in the living body tend to support the opinion that arteries have other terminations. Thus the phenomena of secretion and nutrition prove the distribution of arteries to exhalant surfaces and secreting organs; but whether, as Haller has supposed, the vessels thus ramifying become at once continuous with the secreting duct of a gland, or form the exhalant tube in the surface of a membrane; or whether, as stated by Mascagni, the arteries have lateral pores through which the secreted or exhalant fluid passes directly from the blood—we are, in the mean time, perfectly unable to determine. In like manner, from injections having in some instances been transmitted from arteries into lymphatics, Magendie has espoused the doctrine first promulgated by Bartholine, that there is a direct communication between these two sets of vessels; but the proof of this is not yet complete. Again, the termination of arteries in vessels capable of only transmitting the colourless portion of the blood, rests on such evidence as to make it no longer a matter of uncertainty. When the course of the blood, in the transparent parts of an animal, is watched with the microscope, the vessels through which it passes are seen to become gradually smaller, till at length we can only detect a single globule within their canal. Our means will not permit us to carry the examination farther; but the inference is fair, that there are vessels still more minute which we cannot perceive, and through which the colourless portion of the blood is moving. Nay, we are perfectly assured that many textures are nourished by transparent vessels alone, for the phenomena of acute inflammation and mechanical irritation have shown that such textures may be suddenly distended with red blood; and thus the vascularity of parts, which are colourless in the natural state, is rendered apparent by disease. Setting aside, therefore, part of the hypothetical description given by Haller (for he himself allows, that where direct investigation failed, he had recourse to the uncertain evidence of analogy), we may admit without much fear of future contradiction, that arteries, when traced to their ultimate terminations, either become continuous with a red vein or veins, or pass into colourless vessels, or ramify in secreting organs or textures in a way at present unknown to us.

Founded partly on this view of the minute arteries conveying red or pale blood, Bichat has described the capillary system under three different heads; in organs in which it contains blood, as muscles; in organs in which it contains blood and other fluids, as bone; and in parts in which it contains no blood, as in ligament or tendon. Such an arrangement is quite useless in a practical point of view, it being sufficient to show that parts are either coloured or colourless, according to the size of the vessels with

which they are supplied, and the blood which is circulating through them. To this system of vessels, invisible to the unassisted eye, but directly demonstrated by the aid of the microscope, and indirectly observed through the presence of inflammation, ought we to restrict the term capillary. Under this head also, and with equally good reason, should be included the commencement of the veins; so that we may describe the capillary system as made up of all those minute vessels which are interposed between the arteries and veins, forming the terminations of the one and the commencement of the other. No stricter definition can be given, as it is impossible to say where the arteries end and the veins begin; and, in all probability, the distinctive characters of either are here intimately blended together.

The capillaries have been usually arranged under two sets or orders—those interposed between the arteries and veins of the general system, and those forming the communications between the pulmonary vessels. In the first, the blood is changing from the scarlet colour we find it in the arteries to the modena hue it acquires in the veins; in the second, it is purifying itself by exposure to the atmospheric air, reacquiring its vermilion tint, and along with it those properties which are necessary for the support of animal life. The capillary system is thus at once the most extensive and important in the body. Of the number of vessels included under this name, we cannot form the most remote conception. They exist in myriads wherever organised structure is found, and from the appearance under the microscope, or during the existence of inflammation, we might be almost led to suppose that the human body was one complete net-work of arteries and veins. Of its importance, it is sufficient to notice, that the capillary system is that in which the vital functions of secretion, nutrition, exhalation, and animal heat, are going forward—in which the blood in the general circulation is changed from crimson to dark, and again in the lungs from dark to crimson—in which the phenomena of inflammation more immediately reside—in which the earliest and most important deviations from health, leading to every species of morbid action, might probably be traced—and to which, insignificant as it may at first seem, the larger vessels are entirely subservient.

When a transparent texture in a living animal is placed under the microscope, the capillary circulation is readily brought into view. No definite arrangement of vessels, either as to size, number, direction, or mode of termination, can be perceived. In some of the vessels, three or four globules of blood are moving abreast; others seem only capable of admitting one. Some continue in a straight line; others are reflected back, and terminate immediately in a vein. Some send off lateral branches into veins running parallel to them; in others, one artery ends in a corresponding vein; while again, three or four branches may be observed to be continuous, with only one venous trunk. The capillaries anasto-

mose freely with each other, the veins more so than the arteries; the latter are also smaller and less numerous than the former. Dr. Marshall Hall,[1] who appears to have investigated the anatomy of the capillaries with a wonderful degree of minuteness, has described these vessels as different from either arteries or veins, in as far as they retain a uniform size. According to him, a minute artery is observed finally to divide into two branches, equally large as the trunk from which they proceed, and to anastomose with others of nearly the same magnitude as themselves. To this latter order of vessels, in which the blood moves so much more slowly that its individual globules may be counted, he restricts the name of capillary. The capillaries would, therefore, be vessels of uniform diameter, neither becoming smaller by subdivision, like the arteries, nor larger, by inosculation, like the veins. The arrangement in the lungs is somewhat different, the arteries giving off capillaries from their sides as well as extremities, and the veins receiving them in like manner; neither do the small arteries subdivide so minutely as in the general circulation, but terminate more immediately in capillaries, which are farther distinguished by a more free anastomosis. The uniform diameter which he ascribes to the vessels, has the effect of detaining the blood in the lungs for the more complete exposure of the fluid to the action of the air, and in the different organs for the better performance of their functions. With respect to these observations on the pulmonary capillaries, in the saurian and batrachoid reptiles, to which Dr. Marshall Hall's researches extend, we have nothing to remark; but as regards circulation, either in the web of the frog's foot or the mesentery of the rabbit, we have never been able to observe any thing like the definite arrangement here laid down. It is, however, just to state that, in an investigation where so much deception may exist, no two accounts can be expected to correspond, and Dr. Marshall Hall's means of information may have been better than ours.

In consequence of the extreme minuteness of the capillaries, no correct account can be given of their structure, even were it agreed upon what does and what does not constitute this order of vessels. In observations on the structure and functions of the blood-vessels, contained in the first part of Mr. Hunter's treatise, we find it laid down, that the fibrous or muscular coat is stronger and more distinct in the small arteries than the large; and in proportion as the vessels diminish in size, so in like manner does the middle tunic increase in strength, to enable the blood to be circulated by a power inherent in the arteries themselves, and independent of any direct influence derived from the heart. Every anatomist will willingly admit, that the fibrous tunic is most distinct in arteries at a distance from the centre of the circulation; but the mind must not be led away by this statement to suppose, that the same structure is equally perceptible in the capillaries, because in these no such

[1] Essay on the Circulation of the Blood.

fibres can be detected, nor, in fact, in any vessel less than a line in diameter. The capillary system, although intermediate between the arteries and veins, cannot be regarded as consisting exclusively of either; for the distinctive characters of each are necessarily lost. Bichat conjectured that the internal tunic of the arteries is prolonged to form the ultimate tube of the minute vessels, and thus becomes continuous with the veins; hence he infers, that the common membrane of red blood is identified with the common membrane of black blood. The supposition is natural, that the serous tunic of the arteries and veins should be found in the capillaries, independent of the fact of this membrane being essential to the free movement of the blood along the vessels; but if experiments have shown the extreme branches to be irritable, and if this property of irritability resides only in the middle tunic, then we must allow the capillaries to be invested also with a fibrous structure.

Let us now briefly examine some of the physical and physiological properties of the arteries. In doing so, we need scarcely observe that it is necessary to divest the mind of all previous notions respecting the muscular powers or otherwise of the vessels, and to look calmly and attentively into the phenomena presented to us. The first, though perhaps not the most important, property of arteries is their elasticity, distinguished from all others by remaining equally after death as during life. It resides principally in the outer tunic, partly in the middle, and least, if at all, in the internal. It is much more conspicuous in the large than in the small vessels. When an artery is divided, either in a dead or living subject, its cut ends recede, and may again be made to approach each other; or when a weight is attached to the lower extremity of the vessel, it will be extended lengthways, and then suddenly retract when the extending force is removed. So strongly does this elastic power exist in arteries, that Dr. Gordon applied thirty pounds avoirdupois to a portion of the carotid in a man, before the coats of the vessel gave way. By virtue of their elasticity in the longitudinal direction, arteries restore themselves after being elongated in the ordinary motions of the body, as we see exemplified in those which pass across the cavity of a joint.

Arteries are likewise elastic in the circular or transverse direction, and this may probably be dependent both on the first and second tunics. Elasticity in this way is shown, by the mouth of a divided artery being found open, and always preserving its cylindrical form—circumstances by which we are enabled to distinguish arteries from veins in searching for and tying the vessels in wounded parts. Other proofs of the circular elasticity of arteries are derived from their extension and subsequent retraction in this direction, and from the compression exerted on the finger when introduced into the mouth of an artery so as to distend it. The diameter of arteries is probably greater after death than during life, as their elasticity cannot then be counteracted by the contractions of the fibrous tunic. During the quiescent state of the circulation,

the circular elasticity of the arteries does not seem to be brought into play, at least, to an extent appreciable to the naked eye. Nothing now appears more absurd, than the vast influence attributed at one period to the elastic powers of arteries in carrying on and maintaining the circulation; while how little is to be ascribed to this, under ordinary circumstances, may be shown by a simple experiment. When a large artery, as the carotid in a horse, is exposed, no motion is perceptible so long as the animal remains quiet; nor can the circulation through it be ascertained, unless the finger be applied to the sides of the vessel. Dr. Parry thus first explained the true cause of the arterial pulse, which had been set down by some to the dilatation and contraction of the artery; by others, to an elongation during the systole of the ventricle; and by Bichat, to a change of place in the vessel; but which, in reality, is caused by the compression of the finger obstructing the free transmission of the blood. The phenomenon, however, varies a little, according to the direction of the artery. In the experiment, for example, just mentioned, neither dilatation, contraction, or motion of any kind are visible, while the animal remains free from alarm, and the same may be observed in any other artery which takes a straight direction; but in arteries that are curved, as the temporal, the vessel is moved from its place at each stroke of the heart, so as to allow the pulsations to be distinctly counted. It is evident that the impulse from the current of blood against the curvature of an artery must either cause that portion of the vessel to dilate, or to change its place; and if, in no ordinary case, the former can be observed, the latter must invariably follow. Bichat, therefore, was so far correct in the explanation he gave of the arterial pulse. No violent action of the heart, in the several trials we have made, could effect dilatation of an artery; for when the animal struggles, and the contraction of the ventricle becomes short and rapid, the carotid leaps from its place, and is rendered tortuous.

On comparing these results with what we observe in arteries in the dead subject, it becomes obvious, that, in the living animal, some counteracting force prevents dilatation. This can only be ascribed to a vital contractile power inherent in the middle or fibrous tunic. These two powers are continually opposed to each other during life; the elastic tending to increase the diameter of the artery, the contractile to lessen it; and it is from the full continuance of the former after life is extinct, and the necessary cessation of the latter, that we find the arteries larger after death. In the dead body, the blood is not sufficient to fill the vessels; in the living, on the other hand, the arteries are always full of blood, moving towards the veins. At each stroke of the ventricle the column is moved forwards; and instead of the force of the heart being expended in effecting dilatation of the vessels, it is wholly employed in propelling the fluid onwards. Hence the amount of power economised by arteries not dilating during the circulation of their contents. These observations show that arteries are not

mere elastic tubes, otherwise they would be acted on, and again react, according to the distending force applied. They likewise teach us how little this property of elasticity is concerned in the movement of the blood, and how extremely overrated its influence has been by many physiologists. If we were allowed to form an estimate of its use, we should say that, in the longitudinal direction, elasticity enables arteries to accommodate themselves to the motions of the body; while, in the circular direction, it preserves their cylindrical form, and keeps their canals open, thus opposing the action of the fibrous tunic. It exercises no active influence, however, over the circulation of the blood; for neither dilatation nor contraction are visible, when an artery, in a living animal, is laid bare.

The arterial system displays organic properties totally unallied to elasticity, and the consideration of which is of far more essential importance to the physiologist. These may be distinguished from what we have just investigated; first, by their ceasing shortly after animal life has been extinguished; and, secondly, by their residing solely in the middle tunic. They are ultimately to be resolved into a power of contraction possessed by the transverse fibres of the second coat; the precise nature of which, however, has been a subject of the keenest dispute. By some, these fibres are believed to be muscular, and accordingly they speak of the muscularity of the arteries; by others, who are unwilling to admit an hypothesis which has never been proved, but who yet believe in an active power of contraction, it is called the irritability, tonicity, contractility, or vital force of the arteries; while a third party have described the vessels as mere passive tubes, destitute of every thing save elasticity. We cannot afford space to enter at length upon this controversy, or refer to all the usual leading authorities of the day. We must content ourselves with pointing out simply what we deem the most important particulars.

When describing the second tunic of the arteries, we stated that it was elastic, compact, and easily divided by the pressure of a ligature—that the effect of alcohol, warm fluids, and diluted acids, was to crisp and harden its texture—that no fibrin had been discovered in its composition, according to the best analysis of the present time—and that, for these reasons principally, we are inclined to doubt the identity of its fibres with those of muscle. Dr. Marshall Hall has endeavoured to prove that the arteries are both muscular in structure and irritable in property, but his proofs of the former are unsatisfactory. The experiments, again, of Haller and Bichat have gone to establish the doctrine that the arterial system is destitute of any vital power of contraction, although Haller very singularly espoused the opinion of the irritability of the arteries. Bichat, therefore, if not the earliest, was at least the first successful opponent of the theory of vital contraction. According to his researches, no mechanical or chemical agents, applied as stimuli, are capable of producing any change in the living arterial membrane; neither are contractions visible when

the fibres are dissected off in layers. Opium has no effect in paralysing the fibres of arteries, as it does those of muscles; nor has galvanism any power in causing their contraction. Nysten has since performed some experiments which would seem to countenance the opinions of Bichat. In a few cases of violent death in the human subject, this individual found galvanism insufficient to produce any contraction in the aorta, while the heart could still be made to act; and the same experiment was tried, and with the same effect, on the aorta of a dog. But we cannot receive this as any evidence of the passive condition of arteries; because the power of contraction resides in the fibrous tunic, and the aorta, at its commencement, and for some part of its course, is made up nearly, if not entirely, of elastic substance. In performing experiments with a view to ascertain the properties of the arterial system, sufficient attention has not been bestowed on the difference of structure in the different branches; and thus the peculiarity in the tissue of the aorta not only explains the apparent absence of contractility, but likewise the statement made by Magendie, that this vessel is visibly dilated at each stroke of the ventricle.

The conclusions at which Bichat arrived have been completely overthrown by the experiments of Vershuir, Thomson, Parry, Philip, Hastings, Wedemeyer, and Posieulle, all of whom confirm the opinion embraced by Mr. Hunter, that arteries possess a vital power of contractility inherent in their living fibre. A capability of acting upon their contents—by contracting so as to diminish the diameter of their canal, and again relaxing so as to enlarge it—can be shown to exist in arteries by so many direct experiments, and is so strongly confirmed by other evidence, that no doubt need be longer entertained respecting it, whatever name may be bestowed, or objections raised as to its exact nature. This irritability is displayed by all the branches of the aorta, being still more conspicuous in the small than the large vessels; and it was on account of being endowed with this property, and not from any positive demonstration, that we stated our belief of the capillaries possessing a fibrous tunic. The arteries are under the influence of various stimuli or irritants, such as acids, alkalies, alcohol, the scalpel, galvanism, &c. In applying stimuli to the coats of arteries, it is necessary to draw a line of distinction between those which cause constriction at the expense of their mechanical properties, and those which simply act by inducing contraction of their fibres. We do not think that this distinction has been sufficiently attended to by those who have made experiments on the subject; and it would not require much address to prove that the conclusions to which many have come are on this account alone illegitimate. Thus, when acids and alkalies, or even strong alcohol, are applied to the coats of arteries in living animals, we are told they cause contraction, and that this is sufficient proof of irritability. Contraction, no doubt, follows their application; but the vessel, in most instances, is rendered incapable of again relaxing;

its coats, when examined, are found destroyed; and its tissue crisped and shriveled from the action of these agents on the contained albumen. Hence, such is assuredly no evidence of contraction, which should alternate with, or at least be followed by, relaxation. It is still more important to beware of this fallacy when investigating the properties of the capillaries; for these vessels, from their greater delicacy of structure, are more readily injured. When alcohol, for example, is employed to point out the irritability of the extreme vessels, it will be found that their contraction is not so much owing to the stimulus given by the fluid as to the intense cold caused by its evaporation, and which is often sufficient to coagulate the blood. Dr. Marshall Hall, in experimenting on the web of the frog's foot, has stated that, by the application of alcohol, two distinct layers of capillaries are brought into view; one superficial, in which the motion of the blood is suspended; the other deep seated, in which it still continues to move. The distinction into a superficial and deep layer, does not depend, so far as we have been able to observe, on any such arrangement of the vessels, the motion of the blood being merely retarded in those placed nearest the surface from the effects of cold.

When any of the liquid agents we have mentioned are used in a diluted form, they act like ordinary stimuli, and do not affect the structure of the vessels. Applied in this way, they produce constriction of the artery at the point irritated; which constriction, after remaining for a time, is succeeded by relaxation. Weak volatile alkali, applied to the web of the frog's foot, or the mesentery of the rabbit, causes immediate contraction and paleness of the part, followed by relaxation, turgidity of the vessels, and slower circulation. When the coats of the arteries are irritated with a needle or point of a scalpel, the same effects ensue, only more limited in extent, from the nature of the exciting cause. Similar phenomena may be obtained by the application of galvanism to the capillaries, and the late researches of Dutrochet and Wedemeyer have shown that the contractile power of these vessels is under the immediate influence of this agent. The capability of contracting upon their contents is admirably seen in the capillaries, by watching the changes going forward when the circulation through the larger arteries is interrupted. Thus, when a ligature is drawn tightly round the limb of a frog, or when the aorta has been tied, or the heart itself is cut out and removed, the blood may be observed circulating with little interruption through the capillaries. It was this power of maintaining the circulation after the heart had ceased to act, that Bichat first described under the name of insensible organic contractility; and it is owing to its influence entirely that the smaller arteries are found empty after death. Dr. Marshall Hall has contended that there is no proof that the capillaries are irritable; but herein we beg to differ from him, and to maintain that, independent of other evidence, direct experiments prove the contrary, and these are so readily performed that no suspicion of their validity can be entertained.

The contractile power of arteries may be further illustrated. When irritating fluids are injected into these vessels, in a living animal, great difficulty is experienced in pushing them onwards into the minute branches; while, in the dead subject, the same experiment may be repeated with ease. It must be evident that the resistance in this case cannot be attributed to any other property of the arteries than their power of contraction; for did it depend on elasticity, a bland fluid would be as strongly resisted as an irritating, and both would be equally so after death as during life. The tonic contraction of arteries remains for a short time after an animal has been killed; and when, according to Mr. Hunter, an injection is pushed into the vessels of one newly dead, it is apt to be forced back from the smaller into the larger branches. It is impossible to inject the capillaries immediately after animal life has been put out, as Bichat, Parry, and others, have observed; the injection being only transmitted through the larger branches communicating with veins. These experiments are likewise so many proofs that the contractile power is greater in the small than in the large arteries. When a portion of an artery, removed from a living animal or one newly dead, is distended with water, and then allowed to contract, the amount of reaction is greater than the force used in distension, as measured by the height of a column of mercury the water will support; and this phenomenon may be observed to continue for a short time after death. If a large artery in the living body, as, for instance, the carotid in an ass, or the crural artery of a dog, be rubbed for half a minute, between the finger and thumb, its diameter, at the point so treated, becomes sensibly increased. Upon cutting out a portion containing the dilated part, the whole contracts pretty equally; and on slitting it up longitudinally, the pressure appears to have produced no ecchymosis or injury to the coats of the vessel.[1] When a large artery, as the posterior tibial of a dog, is laid bare and exposed for a time, its diameter, according to Mr. Hunter, becomes so much contracted as almost to obstruct the circulation through it. The contraction of the diameter of an artery is also seen by simply removing a portion of the vessel from the living animal, when it will be found gradually to diminish to half its former size. When an artery is surrounded with a ligature, it contracts both above and below as far as the nearest anastomosing branch. Dr. Parry has farther stated that, if the thread is merely placed round the vessel without being tightened, the latter contracts at the point—where the former lies in contact with it; but retains its original magnitude on either side. The last direct proof of the contractility of arteries which we shall mention, is drawn from the phenomena observed when the vessels are cut across. When an artery, as the radial or ulnar, is wholly divided, its calibre becomes gradually diminished in a circular direction; the stream of blood, which first flowed in jets,

[1] Mayo's Outlines of Physiology.

successively decreases in volume, till at length it only oozes, and then finally stops. When a large vessel, as the carotid, is similarly treated, a slight contraction of its extremity may still be observed; but the impetuous flow of blood speedily proves fatal, and there is not time to watch the changes induced. The contraction and subsequent closure of the mouth of a bleeding artery, arise solely from the action of the transverse fibres, and cannot be explained, as some have asserted, by a languid state of the circulation, or a stronger disposition in the blood to coagulate; in the first place, because these could not produce the effect in question; secondly, because the phenomena are observed prior to the occurrence of either, and where they have been specially guarded against. We are indebted to the late Dr. Jones for illustrating the efficacy of this principle in diminishing the amount of hemorrhage; and it would appear, from his experiments, that it is better seen in some of the lower animals than in man. The vital contraction observed in the extremities of divided arteries, extends throughout the whole arterial system while hemorrhage is going on; and it is from this circumstance that the vessels are enabled to adapt themselves to the quantity of fluid in circulation. Bichat has termed this adaptation *contractilité par defaut d'extension*—in other words, contractility from want of a distending agent; but all attempts to connect it with any other mode of explanation are evidently erroneous, and, in the experiments made both by Mr. Hunter and Dr. Parry, it was found that the arteries in animals dying of hemorrhage contracted to a smaller dimension than that which they maintained after life had been some time extinct.

Besides the direct evidence furnished of a vital or tonic contractility, inherent in the arterial fibre, there are other phenomena occurring in the human body which necessarily bring us to the same conclusion. These we may generally state to be—the remarkable local distributions of blood, and the enlargement of vessels taking place independent of the action of the heart—the effects of agents in promoting or retarding this species of local plethora—and the forces required to carry on the circulation. The instances of local determinations of blood from various causes, both mental and physical, are so familiar to every one as scarcely to require being pointed out. The suffusion of the countenance from shame, the increased flow of saliva from the sight or smell of food during hunger, the redness of the skin from friction, the momentary vascularity of the conjunctiva from the contact of a foreign body, are examples of this description, and all may take place without any alteration in the force or frequency of the heart's action. In a similar manner we find that arteries increase in size and become tortuous. When a ligature is placed round the main artery of a limb, the anastomosing branches immediately enlarge to carry on the circulation. Witness also the tortuous course of the arteries supplying the parts of generation in animals during the season of heat, of those of the arteries during pregnancy, of those

supply organs from which periodical secretions are furnished, or of tissues in which morbid tumours have sprung up. In many cases, also, of local inflammation, and in what are termed passive hemorrhages, increased quantities of blood are transmitted to and contained in parts, where not only is there a want of increase in the heart's action, but where the general circulation, together with the powers of life, is flagging. And we would particularly point out this fact to the attention of the student, because it affords an explanation of a peculiar plan of treatment successfully adopted, and constitutes a most important feature in practical medicine, little appreciated and still less understood. In many of these instances where the distribution of the blood is altered, and determinations made towards local parts, it has been supposed, by Dr. Charles Parry, that there is a prior increased action of the heart. Granting, however, this were true, the general circulation might be thus accelerated, but the greater flow of blood through certain channels could not be accounted for, without admitting the principle that arteries have within themselves a source of local action. The vital contractility of arteries consists in the power which the fibres of the middle tunic possess of shortening and elongating themselves, by which means the diameter of the vessels is increased or diminished so as to admit more or less blood, and by which, also, the circulation of that fluid must be influenced. It is, therefore, an interesting question to enquire, how the coats of the arteries stand affected as to contraction or relaxation? Or, in other words, what is the condition of the artery which gives rise to the determination of blood? The answer to this, however, is so intimately connected with the theory of inflammation, that we defer its consideration till we come to treat of the latter.

The tonic contractility of arteries is under the influence of external and internal agents, the most conspicuous of which are heat and cold, the abstraction of blood, and the emotions of the mind. Heat relaxes the contracted fibres, and thus enlarges the calibre of arteries. We have examples of this in the renewal of hemorrhage from wounds, when warmth is applied; and by the greater fulness of the radial artery, contrasted with that on the opposite side, when the arm is immersed in hot water. Blood drawn from the median basilic vein, under these circumstances, flows more rapidly; and as this rapidity depends on the quantity passing through the artery, it is a strong evidence of the relaxation and enlargement of the vessel. Cold produces, as might be anticipated, opposite effects to those of heat; it increases the contractile power of arteries, and thereby directly diminishes their diameter. When small bleeding vessels, as those in the face of stumps, are spunged with cold water, the hemorrhage ceases. When the arm is held for some time in cold water, the skin shrivels, and the pulse at the wrist becomes smaller. This, performed at the same time with the experiment of plunging the other arm in warm water, enables us to observe better the condition of the radial arteries as to fulness, and the bulk of

the two extremities from the quantity of blood in circulation. When an irritant is applied to a transparent animal texture, so as to cause increased redness, turgidity of the vessels, and slower motion of the blood, bathing the part in cold water makes the vessels contract, and restores the previous paleness.

Abstraction of blood from the system under ordinary circumstances increases the vital contractility of arteries, and by that means keeps the internal arterial tunic in close contact with the circulating mass. This phenomenon is best seen in animals dying of hemorrhage, and is progressive with the loss of blood; it is an important provision in nature for lessening the risk from wounds where arteries are divided, as well as in enabling the vessels effectively to carry on the circulation. We should have supposed *a priori*, that the loss of blood would have the effect of relaxing the arterial fibres, and thus enlarging the diameter of the vessels; but the fact is otherwise, as shown by contraction going on till the mouth of the divided artery is closed—by arteries in animals perishing of hemorrhage acquiring a less dimension than they maintain after life is extinct—and by the necessity for a certain relation subsisting at all times between the size of the vessels and the quantity of fluid in circulation. There are a few instances where the abstraction of blood makes the pulse at the wrist fuller, and which appear an exception to what we have now stated. In inflammation of the serous membrane of the intestines, the pulse, from being small and wiry, becomes soft and full after free depletion. Here the relaxation is probably induced by the relief given to the local disorder, which, taking place in structures supplied from the same nervous source as arteries, has the effect of causing contraction of their fibrous tunic. When more blood is afterwards taken away, the usual changes in the condition of the pulse ensue, and therefore our statement is not invalidated, that abstraction of blood increases the vital contractility of arteries. It is to the ever-varying state of the contractile power of arteries that we trace the different relations of the pulse as to hardness and softness, smallness and fulness; and it is by means of it, coupled with the force of the heart's action, that we estimate the amount of vital power within the system in many cases of disease and decay.

The emotions of the mind influence the tonic contractility of arteries, and the effect varies according to the particular mental change which is present. Thus, shame causes increased redness, as seen in the act of blushing; while fear blanches the countenance, and diminishes the fulness of the pulse at the wrist. We do not stop to enquire on what these opposite states depend; they are referable to the laws which regulate local determinations of blood, and which we mean afterwards to consider. The power of contraction and relaxation inherent in the arterial fibre is without the control of the will, and is therefore to be ranked among the phenomena belonging to organic life. It is still, however, in a general way, under the influence of the brain and nerves, as

witness the effect of mental emotions. It is, moreover, intimately united in sympathy with the heart; for without this consent the circulation could not be steadily carried on. We might point out the connection subsisting between the heart and arteries in a variety of ways; but the details, though extremely interesting, would lead us too far, on the present occasion, into the regions of physiology. The origin and distribution of the nerves supplying the arterial fibres enable us to explain their consent with the heart's action, and why, like this organ, their contraction and relaxation are independent of any immediate impression from the brain or spinal cord.

The evidence now adduced, leaves no room to doubt that arteries possess a power of acting totally distinct from elasticity; and were anything farther required on this subject, we might refer to the indirect proofs furnished by a consideration of the forces required to maintain the circulation. This necessarily leads us to discuss the question, whether the heart is the sole agent engaged in effecting the movement of the blood, or whether any additional assistance is derived from the arteries? The easiest and most certain method of arriving at the truth on this important point, is to contrast the estimated force of the heart's action with the obstacles to be overcome in carrying on the circulation, and to institute a careful examination into the experiments made with a view to ascertain the influence of the capillaries. It is much to be regretted that our information on both these heads is still unsatisfactory, and especially as respects the capillary circulation. The force with which the left ventricle of the heart contracts has been very differently estimated, by some even supposed to exceed a thousand pounds in weight. The most accurate calculations have been furnished by Dr. Hales, although there is reason to believe that they likewise fall short of the truth. This gentleman, after numerous experiments instituted on horses, ascertained the height of the column of blood in a glass tube inserted into the larger arteries, and compared that with the area of the left ventricle in the same animal. He supposes, in the human subject, that the blood in the caroted artery would rise in the tube about $7\frac{1}{2}$ feet; the internal area of the left ventricle being equal to 15 square inches; these multiplied into $7\frac{1}{2}$ feet, would give 1350 cubic inches of blood pressing upon the ventricle when it first begins to contract, and to overcome which would require a weight equal to 51.5 pounds. The first part of this proposition, viz.—that the blood would rise to a certain height above the level of the left ventricle, is a mere hypothesis, for believing in which no evidence can be adduced. We ought to recollect that the heart is a muscular, not an elastic organ, capable of contracting more strongly at one time than at another, receiving likewise more or less blood into its cavities, and enabled from both these circumstances of modifying the distance to which its fluid would be emitted, or rise in a glass tube, especially in an artery so near as the carotid. The contractile power of the vessels would also in-

fluence all such phenomena; for when an opening is made in an artery, a current of blood is established towards it from the anastomosing branches, as well as from those between it and the heart. For these reasons it is quite impossible to determine, with any thing even approaching to accuracy, the force with which the left ventricle of the heart contracts. The calculations of Dr. Hales have been regarded by many as a close approximation to the truth; in our estimation, they are far too high, and we do not think the pressure exerted upon the blood during the systole of the ventricle can exceed a few pounds. Some idea may be formed, as Sir Charles Bell has suggested, of the force with which the heart emits its blood, from the pressure required to stop the current through a large artery in its vicinity, and which may at any time be readily accomplished by the finger. Forming our judgment on this, and on what is witnessed when large arteries are wounded, we are inclined to believe that the blood would not rise one-half the distance which Dr. Hales supposed, and we look upon all that has been written regarding the immense power of the heart, as totally without foundation.

The obstacles, on the other hand, to the circulation of the blood are both great and numerous. They arise chiefly from the elasticity of the arteries, especially those nearest the heart; the viscidity of the blood and its friction along the sides of the vessels; the increasing area of the arterial system and the frequent subdivisions and tortuousness of its branches; the minuteness, reflections, and inosculations of the innumerable capillaries, and the extreme distance of many of these from the seat of power; and the effect of gravity on the ascending column of the blood. The collected amount of these, contrasted with the feeble efforts of the heart, must at once convince us that this organ, unassisted by some other power, would be unable to carry on the circulation. But although the heart alone is insufficient for the general distribution of the blood over the body, there is little question that its impulse reaches to the remotest branches, and influences the motion of the fluid in the most extreme vessels. We prove this by the simple experiment of Magendie, which consisted in tying a ligature round the thigh of a dog, including every part of it except the artery and vein by which the circulation was to be carried on. When an opening was then made in the vein, the blood immediately flowed—pressure being exerted on the artery, the hemorrhage ceased, although the vein was full; the constriction removed, the bleeding recommenced. Nearly the same thing happens on performing venesection at the bend of the arm; for when the bandage, by which the circulation through the superficial veins is arrested, is made so tight as to interrupt the flow through the artery, little or no blood can be obtained. It is stated in some cases where the heart is acting violently, that a pulsation synchronous with, and dependent upon, the stroke of the ventricle, is perceptible in the veins. But we may remark that this phenomenon is only observed in the veins of

the neck, and is probably derived from the contact of the neighbouring arteries; for although the latter have no perceptible motion in ordinary states of the circulation, repeated trials on animals show that they become tortuous and leap from their place when the contractions of the ventricle are short and rapid. If, however, the principle explained in Magendie's experiment is correct, that the flow of blood through the large veins is affected by the *vis a tergo* derived from the heart and arteries, then it is plain that the capillaries, by order of procedure, must be under the same influence; and hence we may infer that the action of the ventricle, although not the sole moving power, is essential to the circulation of the blood throughout the whole of the extreme vessels.

Some idea of the resistance offered to the course of the blood may be conceived from the difficulty with which an injection is forced along in the dead body after all vital contractility has ceased; and so rare is it to effect the filling of the veins from the arteries, by means of the finest injection instead of a thick viscid fluid, that we cannot suppose any power at the commencement of the aorta sufficient to transmit the blood throughout all parts of the body. If, by pressure with the finger on an artery close to the source of the circulation, the force of the left ventricle can be easily counteracted, how are we to imagine that a column of blood, subjected to all the obstacles we have mentioned, can be moved through an almost infinite series of tubes, till it again reaches the right auricle of the heart? But setting aside this sort of evidence altogether, we can show experimentally that the arteries assist in the circulation. When the web of the frog's foot or mesentery of the rabbit is examined with the microscope, the capillary circulation is seen going on for a full hour and upwards after the great vessels about the heart have been tied, or this organ itself removed; and it is from the vital power of contractility remaining for a short time after death that we always find the arteries empty and the veins distended. Were arteries mere elastic tubes, the blood would cease to flow through them as soon as the heart had ceased to act; but that such is not the case is proved by the experiment now related, or by making an opening in the vessels. Another proof of the agency of the arteries in carrying on the circulation is said to be derived from animals without a heart, and of which the imperfect human fœtus is an example; but some doubts are thrown over this by the fact of such a fœtus being always a twin, receiving its blood through the same placenta as the healthy child, and the heart of the latter may thus communicate its impulse to the former.

The arteries, therefore, contract upon their contents, and push the blood onwards into the veins. They seem likewise to have a propulsive action derived from the oblique course which the fibres of the middle tunic take, and by which the column of blood is urged more strongly onwards than could be effected by mere diminution of their canal. The amount of tonic contractility residing in an individual artery would be small; but taken collectively, as

it exists in all the branches, it would, on the other hand, be very great. There is no ground for the assertion, that because pain is not experienced when coats of arteries in living animals are irritated, they are therefore insensible to their contents and incapable of acting. Many other structures are similarly situated as respects external agents, and are only under the influence of their own proper stimuli. The contractility of arteries is excited by the contact of the blood with their lining membrane, in the same way as the movements of the heart are caused by the presence of fluid within its chambers; and, as already stated, both the heart and arteries have the same relation to the nervous system. It is plain also that a perfect consent must exist throughout the whole of the arterial system in propelling the blood onwards into the veins, otherwise the contractions of the lower portion of a vessel would arrest the progress of the fluid in the upper; and contractility being still more conspicuous in the small branches than in the trunks, we conclude that the ramifications of nerves accompany the invisible terminations of arteries. It is from this interrupted supply of nervous energy that we account for all those irregular distributions of blood which vary from the simple act of blushing up to that which constitutes inflammation; and we need seek for no better evidence of the connection between the nervous system and the capillary circulation than the derangement in the function of the secreting and exhaling vessels during the presence of fever.

From what has now been advanced respecting the circulation, it would appear that the heart and arteries are equally engaged in carrying on this important function. Without the one, the blood could not be transmitted in successive jets, nor that *vis a tergo* derived which seems essential to its motions in the veins; without the other, the contents of the vessels could not be conveyed to the most distant parts of the body, and again brought back to the chest. We are not, therefore, among the number of those who consider that the blood, as it passes through the capillaries, is beyond the influence of the heart, oscillating backwards and forwards, and at length moving on in the only direction left it. No such wavering motion can be perceived by the microscope, neither can the individual globules of the circulating mass be distinguished, unless some cause of stagnation is present. The impulse of the heart, however, is much diminished before the blood reaches the extreme vessels. The motion of the fluid, as it traverses the capillaries, is more continuous than in the branches immediately before, and viewed through the microscope in the transparent part of an animal, the blood visibly flows in one continued stream from the arteries into the veins. The motion of the blood through the capillaries has been supposed to depend on the difference in the electrical relations of the fluid in the arteries and veins; but we have no right to explain a natural fact by the admission of a principal of which we have no proof, and however satisfactorily this might account for the systematic circulation, where the blood is changing from scarlet

to dark, it would not apply to that through the lungs, where the phenomena are exactly reversed. Neither will the attraction of fluids of different densities for each other through porous substances, as so ably investigated by Dutrochet, explain the motion of the blood in vessels endowed with a vital power of contractility, and having a separate independent action of their own. All attempts to illustrate the intricate operations of life, by referring them to physical laws, are both delusive and hopeless.

It is impossible to determine accurately the velocity with which the blood moves in the different vessels. We neither know the exact amount in circulation, nor how much is emitted at each contraction of the ventricle; and if even both were ascertained, the variation in the size of the different vessels, and that, too, at different periods, would leave us still at a loss to decide. In considering this point, the student will do well to remember the difference between the mean velocity of a single pulsation and that of the column of blood. The arteries in the living body are constantly full during the ordinary state of the circulation, the fluid within them receiving a distinct impulse from each fresh portion thrown out at the systole of the ventricle. The blood at first moving in jets through the larger arteries gradually loses its projectile motion, till at length in the capillaries it flows in one continuous stream. The impulse communicated to the column of blood by each successive portion as it leaves the heart, travels, according to the calculations of Dr. Young, at the rate of sixteen feet in a second, nearly three times the length of the body in a person of short stature; the beat of the pulse should, therefore, appear synchronous with the action of the ventricle, and the throb of the arteries be simultaneous over the body. The velocity of a pulsation, however, gradually diminishes as the distance increases; and hence a distinct interval of time elapses between the impulses of the heart against the ribs, and the beat of the artery at the wrist. If, in a living dog, a small puncture is made in the ventricle, and another in the main artery of a hind extremity, a distinct interval is perceived between the jets of blood. We have seen it repeatedly stated, in works, too, of considerable merit, that the arteries in one part of the body may have their pulsation increased in frequency beyond that of others, or even of the heart itself—as the radial artery in whitlow beating more quickly than that of the opposite arm. We need scarcely observe that this is a great error; the impulse given to the blood is derived from the heart and not from the arteries, and therefore the pulsation cannot be more frequent in one part of the body than in another.

The mean velocity of the column of blood is much less than the impulse transmitted through it; being, in the aorta, according to the authority lately quoted, about 8 inches in a second. The calculation has been made by taking the average quantity of blood in an adult to be 40 pounds, and supposing from one to two ounces thrown out by each contraction of the ventricle, at the rate of 120

feet in a minute. The contraction of the ventricle, however, only occupies a third of the time from one complete systole to another, and therefore 120 divided by 3 gives 40 feet in a minute; this again multiplied by 12, to reduce it to inches, and then divided by 60, gives 8 inches in a second as the velocity of the blood's motion in the aorta. The mean velocity of a pulsation would accordingly be 16 feet in a second, while that of the column of blood would be 8 inches only in the same space of time. But the arteries form a number of descending cylinders, the combined areas of which exceed those of the trunks from which they are derived. In a state of heath, therefore, while the vascular system is uniformly distended, no congestion present in any part, and the arteries transmitting the whole quantity of blood received by them into the veins, it follows that the velocity of the fluid in the extreme branches will be less than in the aorta, just in proportion as the sum of the combined areas of the former exceeds that of the latter. For the same reason, namely, increased capacity, the motion of the blood in the veins is slower than in the arteries, and in the branches of the veins, than in either of the *venæ cavæ*. The velocity of the blood's motion is extremely liable to variation, as whatever tends to diminish or increase the relative area of the vessels must exert on it a corresponding change. This is a subject of the highest importance in a pathological point of view, and one which seems to be greatly misunderstood. Much valuable knowledge would accrue from a careful investigation into the irregularities which happen in the vascular system, and the provisions made by nature to avert their bad effects.

SECTION II.

THEORY OF INFLAMMATION.

To establish a just theory of inflammation is a point of the highest importance in medical science, as tending directly to improve our knowledge of morbid action, and enabling us to lay down precise rules of treatment, free from all appearance of empiricism. This is a subject still involved in dispute, and one on which even experimental research has failed to produce conviction. We enter upon it, therefore, more with the view of recording the phenomena we have observed, and making up our history of the disease, than with any hope of assisting in the adjustment of so intricate a problem. The difficulties we have to contend with, in watching the circulation in a state of health, apply themselves with double force when investigating the nature of inflammatory action; and were our acquaintance with the former more enlarged, our knowledge

of the latter would be comparatively easy. The more immediate phenomena of inflammation are confined to the minute blood-vessels, and to the changes going forward in them, our attention must therefore be first directed. There is one way only of arriving at the truth, and that is, by inducing the disease in the transparent parts of an animal, observing with the microscope the changes which ensue, and marking carefully the order of their occurrence. Nothing satisfactory can be elicited by considering the symptoms during life, or examining the alteration of structure left after death. On neither of these two heads can we reason correctly. From the first, any conclusions as to the state of the circulation must be vague and uncertain. From the second still less can be gathered respecting the process of inflammation, because its effects alone are visible; the morbid action which led to them has ceased with life.

Two theories of inflammation have long occupied the attention of pathologists—the one setting forth that the action of the vessels is increased; the other that it is diminished—and both of these have been warmly supported. We are of opinion that the question has been two exclusively confined to a mere consideration of the state of the vessels; and that many essential parts of the process have, on this account, been overlooked. In proof of this, we assert that more valuable information has been lately contributed by Gendrin and Kaltenbrunner than by all who have preceded them; and we are especially indebted to the last mentioned writer, whose observations have been limited to the changes presented in a part labouring under inflammation, without having entered into any hypothetical discussion as to the state of the vessels. And had those making experiments not looked too narrowly into the points at issue, and endeavoured only to combat one opinion for the sake of establishing another of their own, we should long ere now have had a more just and satisfactory account of inflammatory action.

Two or three positions respecting the state of an inflamed part have been universally admitted. First, that it contains more blood than one in a natural condition; secondly, that its vessels are distended or dilated; and, thirdly, that minute branches which only previously allowed entrance to a colourless fluid, now contain coloured globules. The controversy has been, whether the vessels have their action increased, and are transmitting more blood through them in a given time; or whether their action is diminished, and there is a stagnation of their contents. When the circulation of the blood was unknown, and the old doctrines of the liver preparing and sending forth this fluid believed in, physicians were so satisfied of the existence of humours and spirits, and of the possibility of blood and bile being poured out or pent up in different parts of the body, that the occurrence of inflammation was easily explained. The peculiar kind of disease was supposed to depend upon the kind of humour prevailing; thus, blood gave rise to phlegmon, and bile to inflammation of the skin. They conceived

that the blood and the different humours might stagnate simply for want of an expulsive power, and to this they gave the name of congestion; while, if there was a sudden rush of humours to a distant part, it was called a defluxion. These rude notions prevailed in the schools till the time of Boerhaave, who constructed a new theory of his own, and supported it with all the learning and ingenuity of which he was so eminently possessed. The diameters of the blood-vessels and of the globules of blood were attempted to be measured, and calculations were made as to their relative fitness for each other. The application of mechanical philosophy to the science of medicine was then a favourite study; and Boerhaave conjectured that particular vessels were set apart for the reception of certain globules. His theory of inflammation was remarkably simple. If a globule was pushed into a vessel too small for its reception, it naturally obstructed the blood behind it; and, to this accidental occurrence, he gave the expressive name of *error loci*. He farther considered that the properties of the blood were altered —that its gluten (now the coagulating lymph) was increased in quantity, while its thinner parts were drained off by the occurrence of diarrhœa, sweating, and the like. The fluid thus rendered more viscid and tenacious, obstruction of the vessels readily ensued. The cause of obstruction, and likewise that of inflammation being discovered, the treatment was to be conducted on the principle of thinning the mass of blood by means of substances termed attenuants, and which consisted chiefly of the alkalies, mercury, and some of the neutral salts. We cannot pay greater homage to Boerhaave than acknowledge, that the fundamental part of his doctrine is borne out by the investigations of the present day; we see his *error loci* and the changes induced on the blood in every instance of inflammation—the manner in which he explained them is alone fanciful.

The mechanical theory of Boerhaave, as well as the humoral pathology which had preceded it, gradually fell before the more stately and substantial system of the solidists, from among whom sprung the celebrated Cullen, who, revising the doctrine of Stahl and Hoffmann, applied them, in a greater state of refinement, to the explanation of disease. The obstruction of the blood which occurs in inflammation was attributed to a spasm of the extreme vessels, to overcome which the use of nauseating doses of antimony and ipecacuan was enjoined. We shall examine this hypothesis at more length when treating of fever; suffice it at present to remark, that no such obstruction is observed at the commencement of inflammation, nor is there any proof of the extreme branches being affected with spasm.

An increased action of the vessels of an inflamed part, was first suggested by Gorter, a disciple of the famous Stahl. He considered the proximate cause of inflammation as an increased vital action of the arteries, whereby the blood is driven with greater force into the colourless vessels. When this increased action took

place in all the vessels of the body, it produced fever; when only limited to a few, inflammation was the consequence. The doctrine of an increased action of the vessels has been advocated by the late Dr. Parry of Bath, by Mr. James of Exeter, and by several who have written detached papers on this subject. It has been said that Mr. Hunter entertained similar views; but his opinion on the nature of inflammatory action is not clearly stated; nor does it appear from his writings that he had ever made any experiments in order to ascertain the state of the minute vessels. We find him calling the act of inflammation an increased action of the vessels, which at first consists simply in an increase or distension beyond their natural size. "This increase seems to depend upon a diminution of the muscular powers of the vessels, at the same time that the elastic power of the artery must be dilated in the same proportion. This is, therefore, something more than simply a common relaxation, we must suppose it an action in the parts to produce an increase of size for a particular purpose." It is difficult either to comprehend or affix any precise meaning to such language; and without pursuing the analysis, we think it evident that this great man found himself involved in a serious dilemma, and framed the best hypothesis he could to make his escape.

In modern times, the vague phrase of increased action has been considered by many to denote all the changes induced in the capillary vessels, and quite adequate therefore to explain the process of inflammation. It would be satisfactory, before going farther, to have a more correct definition of what increased action means, for every affection of the vessels so named does not certainly amount to inflammation. The supporters of this doctrine have founded their views on different circumstances; and first, on the augmented strength in the pulsation of the arteries leading to the affected part. When the hand is the seat of inflammatory action, it is well known that the radial artery pulsates with greater strength than that on the opposite arm; nay, some have even gone the length of stating, that the pulsations are not only stronger but more frequent—an error to which we alluded in our remarks on the circulation, If the augmented force with which the principal arteries pulsate extended throughout the minute branches, then a state of increased action must necessarily be present; but this is not the case. We will allow that the hypothesis is so far correct; but we may ask, does this increased activity in the whole series of vessels remain unchanged during an ordinary attack of acute inflammation? Does the vital contractility of the capillaries enable them to withstand the increased quantity of blood thrown into them from the larger arteries? Or is there any probability that their tonic power is ultimately overcome, and that they are incapable of withstanding the augmented pressure from behind? It is no argument, because the arteries leading to the seat of disease are acting vigorously, that their delicate capillary terminations must be in the same state. On the contrary, we will presently show that the increased activity of

the former leads, sooner or later, to a loss of power in the latter, and a stagnation of the fluid within their canals. No information can evidently be derived from reasoning such as this; for wherever speculation usurps the place of experiment and observation, we only pursue the shadow and lose the substance.

Again, we are told that a sense of throbbing experienced in some cases, as in whitlow, is a proof of the increased activity of the vessels. This is, without exception, the most unfortunate argument that could have been brought forward; and, in place of being a sign of an increased circulation through an inflamed part, it is an obvious mark of obstruction. The phenomenon arises from the resistance the blood meets with in its passage through the smaller vessels, and may be produced at any time by tying a ligature so tight round a finger as to obstruct the circulation. Besides, as Dr. Thomson has well observed, this throbbing or pulsatory motion can afford no proof of the force with which the artery contracts; for it is caused in the dilatation of the artery, and by a power foreign to the artery itself. Another circumstance, on which the doctrine of increased action rests, is, that if the artery leading to an inflamed part be divided, as the digital branch in whitlow, the blood will be ejected to a greater distance and flow more impetuously than from a vessel supplying a similar healthy part.

We believe that every practical surgeon will acknowledge the truth of this remark; but although an increased activity in the larger arteries is thereby distinctly shown, it does not follow that those at the seat of disease are similarly affected, and our objections here are the same as those already offered. It is no proof of the state of the circulation through an inflamed part that it contains more blood than one in a sound condition, for the contending parties admit this point without dispute; nor is it any evidence of either one state or another, because the blood flows more freely when an incision is made; for were the vessels distended from whatever cause, the same thing would result on their division. A careful examination into the venous circulation of an inflamed part, would prove a more powerful argument either for against the doctrine of an increased action of the vessels. During health, the powers by which the blood is kept in motion are so exactly balanced, that no accumulation or congestion occurs in any part of the body, and the quantity of fluid delivered into the veins is precisely equal to that received by the arteries. Had we no better means therefore of judging, the question of an increased or diminished activity of the vessels might be determined by the quantity of blood found returning from the veins of an inflamed texture. If the circulation through the capillaries was much accelerated, the amount of blood delivered into the veins should be greater than in health; but, on the other hand, if the extreme arteries are relaxed, and the fluid stagnating within them, then the incipient veins immediately leading from the seat of disease should be comparatively empty. Now the truth is, that in the earlier stages of inflammation, the circulation is more

rapid—the capillaries are acting with increased vigour—the flow of blood through the part is greater than in health—and the quantity delivered into the veins is consequently augmented; but, in the latter stages, from the motionless state of the blood, as seen by the microscope, the phenomena of the venous circulation must be directly reversed. It is to be regretted that we have no accurate account of the condition of the veins leading from an inflamed part, and especially during the different stages of the disorder, nor did the idea of examination suggest itself till we had nearly concluded our experiments on this subject. When the hand is the seat of disease, and a vein at the bend of the arm is opened, the blood flows more freely than usual; but this, although a proof that more blood is sent to the extremity, is no testimony either of the state of the circulation through the inflamed part, or the plenitude of the veins communicating with the affected capillaries. Where inflammation was limited, no difference in the state of the veins might be observed; where the morbid action again was more extensive, the pressure from effusion would have the effect of interrupting the return of the blood by one set of veins, and of causing greater distension of others. All we contend for is, that there is no evidence by which we can show that, during the existence of inflammation in a part, a greater quantity of blood is continually delivered into the incipient veins, and by which a free and increased circulation through the extreme arteries would be going on.

No conclusion can be drawn, as the advocates for an increased action of the vessels have supposed, from the circumstances of pressure on an inflamed spot causing the redness to disappear, and a momentary paleness to ensue. This can only be observed in an early stage, and where a superficial part as the skin is affected. We can conceive that pressure would as easily remove the stagnant blood out of a dilated vessel as prevent its influx; while, in either case, the action of the larger arteries behind will speedily lead to the restoration of the lost colour. The exciting causes of inflammation are all of them said to be of a stimulating nature, and therefore to prove increased activity of the vessels. These do not, in general, remain long in operation; hence, whatever effect they may at first exercise on the capillaries, must ultimately cease, leaving no ground for the assertion, that one and the same state of action is present throughout. Moreover, it would be necessary to know what amount of stimulus is required, and for what length of time it must be applied; for the exciting causes differ so much in their nature and intensity, that it is difficult to draw a line of distinction between those which exalt and those which depress vital action. Whoever takes the trouble of looking into the phenomena of inflammation as arising from a local exciting cause, will soon perceive the great changes which are induced, not only in the part itself, but in the system at large; and by which new modes of action and new pathological states are developed. So long only as the respective balance between all the vessels is maintained, does no marked

derangement occur in the circulation; and as soon as that balance is subverted, a new series of changes immediately begin.

The doctrine of increased action of the vessels only implies a greater activity in the circulation, by which more blood shall be transmitted through a part in a given time; but during which, according to the ordinary laws of the animal economy, there shall be no accumulation or congestion. We have several reasons for believing that a state of increased action, by which more blood is passing through a part, and where there is no stagnation, cannot make up the whole act of inflammation. We have many instances of local determinations of blood to particular parts, by which the circulation is much increased; but which neither give rise to morbid phenomena, nor have been considered as any deviation from the state of health. Did inflammation merely consist in an increased circulation of blood, it would certainly be a difficult matter for us to conceive to what extent that should be which, on the one hand, led to the most innocent results; and, on the other, to the most serious and destructive changes. We have plenty of evidence to show that, while local determinations of blood may be removed without farther change either to the part itself or to the system at large—such states frequently precede morbid action, and under all circumstances are favourable to its development; and that where the exciting cause which produced the effect in question is allowed to remain, or acquires an increase in power, inflammation may be speedily manifested—nay, more, we have abundant reason to believe that this is the way in which inflammation generally originates; for after disordered action is present, we often find it spontaneously ceasing on the physical cause being withdrawn, and without any other result than merely accelerated circulation. We observe with the miscroscope that an increase in the natural circulation is amongst the earliest effects of the application of a stimulus—that the arteries contract with greater vigour, and propel their blood onwards into the veins; but that although more blood is thus passing through a part, yet no more is contained at one given time, and there are no signs of stagnation. In the same way we are informed that the period of incubation—if we may so name the incipient stage of inflammation—may pass away, the augmented circulation decline, and the part assume its natural appearance; while, if the exciting cause continues in operation, and the morbid action goes on to increase in severity, the healthy equilibrium among the vessels is subverted, the *vis a tergo* increases, the contractility of the extreme branches is weakened, and a stagnation of the circulating fluid becomes unavoidable. The first stage of inflammation consists in an increased velocity of the blood through the vessels, which greatly resembles, and may arise out of, that state to which we give the name of local determination; but such falls vastly short of the phenomena to be observed during the continuance of a morbid action far more complex in its nature. To those who view the capillaries as inert tubes, destitute of all con-

tractile power, incable of exerting any change on their relative capacity, and without any control over the fluid which circulates through them, no other idea of inflammation, save that of increased action, could well suggest itself; but as these vessels can, by direct experiment, be shown to be susceptible of contraction and relaxation, such notions must, for many reasons, be erroneous. We have commented on the ordinary proofs for an increased activity of the vessels in an inflamed part, in order to point out what share of credit the doctrine is entitled to receive. Our objection is, that it has been made too exclusive, and that those, its advocates, have remained content with knowing only the first series of changes going forward.

There is another class of individuals who have given to inflammation a totally different character. They have described the vital energy of the capillaries as diminished, and the blood stagnant in the relaxed and dilated vessels. It being granted that the extreme arteries are the seat of disease, and that, during its presence, they are much dilated, as evinced by the turgidity of the colourless branches, it has been inferred, that increased dilatation and injection with red blood could only arise from relaxation and debility, followed by an obstruction to the circulation. Such seems to have been the opinion of Vacca Berlinghieri of Pisa, who published a treatise at Florence in 1765, wherein he has the merit of promulgating the doctrine of diminished action of the vessels in inflammation. The same theory was afterwards embraced by Mr. Allen, a lecturer on physiology at Edinburgh, and who was probably its first advocate in this country. The difficulty of accounting for the state of dilatation into which the capillaries are thrown, coupled with an increase in their active power of contraction, has led to the opinion that inflammation consists in relaxation and debility of the minute vessels. Were the arteries muscular, or at least endowed with a power of contracting upon their contents, it is argued that they could not be dilated, and at the same time assume an increased activity; because increased action implies increased contraction, which, instead of enlarging, must lessen the diameter of a vessel. When the muscular fibres of the heart or bladder contract, the cavity of these organs is lessened; after the same manner the circular fibres of the intestines diminish their canal and assist in propelling the contents onwards. How then are we to conceive that the contractile power of an artery is augmented, and by which the blood is more rapidly transmitted, while the calibre of the vessel is plainly enlarged? According to the theory of diminished action, the exciting causes, although they may be said to stimulate the vessels, do not produce increased activity of the capillaries. They may rouse the action of the larger arteries leading to the inflamed spot, and ultimately quicken that of the heart itself; but the effect of this inverted order is only to propel an unusual quantity of blood into the part, and especially into the colourless branches, and by which their canals are obstructed. The stagnation thus created, proves that the smaller vessels, instead of having their action in-

creased, are debilitated, and unable to carry forward their contents. The curative influence of the loss of blood will be, to lessen the impetus of the circulation and the pressure from behind; while the topical application of cold or astringents will assist in the contraction of the arterial tunics, and thereby in the expulsion of the stagnant fluid; and it is not a little curious, how plausible and natural the explanation of remedial measures may be, according to either method of reasoning.

The abstract consideration of both theories involves the doctrine of the vital contractility of the extreme branches—their independent power of altering the distribution of the blood, and of accelerating its motion. Unless we grant these, it will be impossible ever to illustrate the nature of inflammatory action. To ascertain the condition of the blood vessels is, of course, the first step towards the completion of our knowledge; although, as we have already remarked, the disease in question is made up of a series of changes exceedingly complex in their details. It has been ingeniously attempted to resolve the local action of arteries, as seen in blushing, &c., into a relaxation of their tunics, and by which an additional effect would be given to the impulse of the blood as it is emitted from the heart. "We have but to suppose that the usual resistance of the vessels to the flow of blood is diminished by a relaxation of their tunics—the final cause of which would be as obvious as the physical cause which determines it is obscure."* According to this explanation, all determinations of blood, whether natural or morbid, towards particular parts, would be caused by the spontaneous relaxation of the coats of the arteries; and the heart, although its action was not increased, would, from a diminution of the usual resistance, be enabled to transmit the blood in greater quantity. If we can once reconcile ourselves to the theory that the coats of vessels are in this relaxed condition, we shall find no difficulty in accounting for the more copious draught of blood determined to particular parts; and all doubts as to the alleged inconsistency of an artery being in a state of increased activity, while, at the same time, its calibre is enlarged, will be completely removed. There are, however, various reasons for our not adopting the opinion, that the relaxation of the coats of arteries is a source of local action. First, the act of blushing is so instantaneous, that, did it arise from relaxarion of the capillary branches, it is extremely improbable that it should cease so suddenly as is observed; for relaxation implies a quiescent condition of the vessels with slower motion of the blood. Secondly, the obstacles to the circulation of the blood are so numerous, that the heart is unable to carry it on independent of a tonic contractile power exerted by the minute vessels; and there being no appearance of congestion in organs to which larger quantities of blood are sent, but, on the contrary, evidence often of an increased nutrition, as in the uterus during pregnancy, we cannot suppose

[1] See Mayo's Outlines of Physiology.

that the capillary system is in a relaxed and passive condition. Thirdly, in most of the local determinations which take place from natural causes, and in many cases of inflammation also, the action of the heart is not increased; and it is therefore impossible to believe that the impetus of the blood from the ventricle could acquire such additional effect from the relaxation of the arteries, as to pass through the extreme branches with the rapidity and in the quantity we witness. Fourthly, the velocity of the blood through the capillaries in the act of blushing is much greater than at the source of the circulation, and consequently must depend on increased contraction, and not on relaxation of the vessels. Fifthly, if local determinations arose from relaxation of the arterial tunics, the blood in the capillaries should have an intermittent motion, being under the more immediate influence of the heart; but no such undulating movement can be perceived. Sixthly, causes, such as fear, which weaken vital action, and should produce relaxation, are not followed by local congestion but the reverse; while those which stimulate or excite, give rise to the phenomena we are now considering.

For these reasons we are not inclined to receive the hypothesis which ascribes local determinations to the relaxation of the vessels, and by which it has been said additional effect may be given to the impulse from the heart; and we adhere to the opinion that all such irregularities are, in the first instance, to be explained by an increased action of the arteries and the power which they possess of altering the distribution of the blood. Neither can we admit, because the extreme branches are dilated beyond their ordinary size, that they must necessarily be in a state of relaxation or debility. Increased action, no doubt, implies increased contraction, and this may be supposed in turn to diminish the diameter of the vessels, and allow of less blood within their canals; but there is evidence to show that, within certain limits, arteries may be dilated so as to accommodate themselves to a greater quantity of blood, and yet preserve their entire control over its motion. The analogy borrowed from the contraction of the fibrous structures is an argument in favour of this opinion; thus, the heart and bladder act more powerfully when moderately distended, and the intestines and uterus in like manner. If we admit that arteries are endowed with a vital contractility by which their inner coat is always kept in close contact with the blood, then we must allow that, within certain healthy limits, they are also capable of distension; for the circulating fluid varies in quantity at different periods, and it is only when these limits are exceeded, either by loss of tonicity in the vessels, or by the pressure of a greater column of blood than they are able to sustain, that relaxation and debility ensue. The microscopical observations we are enabled to make on the early stages of inflammation, point out to us most clearly that the minute branches have their diameter enlarged and their action increased; the hypothesis, therefore, of relaxation being a source of local action in arteries, or inflammation being throughout a state of congestion, arising

from the entrance of blood into enlarged and enfeebled capillaries, is contrary both to reasoning and known facts.

The first accurately detailed experiments concerning the state of the vessels in inflammation were made by Dr. Wilson Philip, who came to the conclusion that the action of the capillaries was diminished, while that of the larger arteries, leading to the seat of disease, was increased. "Thus far," says he, "I cannot help thinking the nature of inflammation appears sufficiently evident. The motion of the blood is retarded in the capillaries in consequence of the debility induced in them; an unusual obstacle is thus opposed to its motion in the arteries preceding them in the course of the circulation, which are thus excited to increased action." He then proceeds to enumerate several difficulties connected with the subject on which his experiments have thrown no light, and which remain unexplained to the present day. Such are the effects produced on the constitution by inflammation in different situations, or the manner in which the disease spreads to contiguous and distant parts—all of which are referable to the agency of the nervous system. He considers that the irritation of the nerves of the inflamed part may excite the larger arteries of this part, or of distant parts, or of the whole sanguiferous system; and that it will, of course, be most apt to do so where the irritation excited by the inflammation is greatest, and consequently in the more important and vital parts. Nor is it a little in favour of the doctrine of a diminished action of the vessels being the proximate cause of inflammation, that its supporters have founded their views on actual observation; while those who have entertained an opposite opinion have not, so far as we are aware, made a single experiment to corroborate their assertions. We have the same objections to Dr. W. Philip's views as to some others which have been brought forward on the same side—namely, that they are too limited, and do not include an account of the phenomena which are seen during the earlier stages.

In order to ascertain the state of the blood-vessels in inflammation, Dr. Thomson instituted a number of experiments in the autumn of 1809, the results of which were detailed in his valuable lectures, published in 1813. These trials were made on the web of the frog's foot; and it is to be regretted they did not include the warm-blooded animals, as it has been objected that analogies between the higher and lower orders of animals cannot be deemed conclusive. These objections have been done away with by experiments since made on a more extensive scale, and which allow us to give full weight to the statements of Dr. Thomson. This gentleman applied irritation to the small arteries of the web, by means of a fine needle, strong spirits of wine, and tincture of opium, all of which, in various instances, increased the general circulation. Weak volatile alkali had the effect of inducing contraction in the arteries to which it was applied, followed by a diminution in the velocity of the circulation through the capillary vessels with which the contracting artery communicated. When

the contraction was complete, a temporary stagnation was observed in the capillaries immediately communicating with the contracted artery, and the corresponding veins were obviously diminished in size. In the experiments with the ammonia, a paleness, rather than a redness, was produced; but this lasted only for a short time. "These observations," says the learned professor, "which I have just related, prove undeniably, I conceive, the existence of irritability in the smaller or capillary vessels of cold-blooded animals; and, consequently, the possibility of irregular distributions of the blood in particular parts of the body being produced, independenty of the heart, by the vital, contractile, or irritable power inherent in even the minutest branches of the arterial system." The most remarkable effects, however, resulted from the application of a saturated solution of common salt, and which varied in different animals, and in the same animal under different circumstances. These may generally be stated as—*increased velocity of the blood, with a sensible enlargement of the arterial and venous trunks*, but after repeated application, followed by retarded capillary circulation, or even by complete stagnation—an apparent increase of the circulation in the arteries and veins, with a diminished velocity in the capillaries, seemingly arising from want of power—most frequently diminished rapidity of the circulation in arteries, veins, and capillaries, the diameter of each set of vessels being enlarged, and the redness of a darker colour than that which is accompanied with an increased capillary circulation. In all the experiments with the salt, the blood-vessels were dilated, whether the circulation through the capillaries was increased, diminished, or altogether suspended. Considering these effects—which lasted for different periods of time, according to circumstances, especially the weakness of the animal and the frequency in the repetition of the salt—as analogous to inflammation, Dr. Thomson infers, first, that the velocity of the blood, so far from being always diminished in inflamed vessels, is often increased, particularly in the commencement of inflammation; and that this increase of velocity may continue in the capillary vessels from the commencement to the termination of that state. This increased circulation occurs, he is inclined to believe, in a greater or less degree, in that state which has been denominated active inflammation. Secondly, he considers that a diminished velocity in the circulation through the inflamed capillary vessels may take place in the very commencement of inflammation, and may continue during the existence and progress of that state. Thirdly, that this diminished circulation in the inflamed capillary vessels takes place, however, more frequently during the progress than at the commencement of inflammation in healthy and strong persons; and that it is probably a state which occurs in those inflammations which have been denominated passive. This inference, he thinks, is warranted by the diminution of velocity produced in the arterial branches by repeated applications of salt, or even in weaker animals by a single appli-

cation. If these views of the circulation in inflamed vessels be just, it will follow, that inflammation is sometimes attended by an increased, and at others by a diminished, velocity through the capillaries of the inflamed part; and, consequently, that neither of these two states ought to be included in the definition we give of inflammation. In a previous part of the same chapter, the author states his belief that a diminished velocity of the blood in the capillary branches is by no means a necessary, constant, nor even the most common effect of *incipient and moderate degrees of inflammation.*

Dr. Thomson has very generally been ranked amongst those who support the theory of a diminished action of the vessels as the proximate cause of inflammation; but how far this can be maintained, we may judge from the quotations now made from his writings. He considers that inflammation may consist in a state of increased action of the vessels throughout, and this more particularly when the disease has assumed an active character. Again, that there may be from the commencement to the termination, a diminished motion of the blood through the capillaries, and that this occurs more frequently during the progress of inflammation in healthy subjects, and in that form which has been named passive. It appears, however, extremely illogical, and even a contradiction in terms, to argue that acute inflammation, during its whole course, should be attended at one time by an increased, and at another by a diminished circulation through the capillaries; or that two such opposite states should make up by mere chance, one and the same species of morbid action. We can show, to a demonstration, that active inflammation is first a state of increased, and then one of diminished action of the vessels—that at its commencement the velocity of the circulation is greater, but subsequently becomes less, and nearly, if not altogether ceases; and that retardation, not acceleration of the blood's motion, is the most conspicuous phenomenon. Neither has it been proved that passive inflammation alone consists of diminished or obstructed circulation through the capillaries of an inflamed part, although considering its being frequently a termination of that which has been acute, the apparently sluggish condition of the vessels, and the effects of remedies, there is reason to believe that the extreme branches are in a relaxed and weakened condition.

Dr. Thomson has drawn his inferences respecting the state of the blood-vessels, exclusively from the appearances presented to him in the web of the frog's foot by the application of salt. This appears to us the principal error committed, and accounts for the opposite and contradictory conclusions at which he arrived; for had he made trial of other means to effect his purpose, he must have witnessed very different results. Common salt, for example, produces instant dilatation of the small vessels to which it is applied, while oil of turpentine removes it by causing contraction; and it would be as unfair to insist, from the use of the latter, that inflam-

mation consisted of nothing but increased action, as it would be to argue the contrary, because retardation of the blood's motion generally follows the application of the former. It is highly essential, in an investigation like this, that we should be aware that different agents produce very different effects; that some cause contraction of the vessels of momentary paleness followed by redness, increased circulation, and ultimately retardation; others, dilatation with quicker and then slower motion of the blood; others, at once complete stagnation; and others again, contraction and increased activity without being succeeded by other changes. Several of these may be made to produce opposite effects according to the state of intensity in which they are applied; and not unfrequently the destruction of the arterial tunics has been mistaken for the influence of stimuli on the vessels. It must appear plain, therefore, how very cautious we ought to be in drawing conclusions from the effects of agents between which so many wide differences exist; and how liable we are to be deceived by confining our view solely to the appearances presented by only one or two. And, while dwelling on this point, we may observe, that by far the most accurate information respecting the state of the vessels is derived from watching the inflammation set up in parts by severe mechanical irritation or violence, as in this way we are free from the deception attending the application of peculiar substances.

Dr. Hastings has more recently investigated the process of inflammation experimentally, and entertains nearly the same views as Dr. W. Philip. He infers, from his researches, that inflammation consists in a weakened action of the capillaries, by which the equilibrium between the larger and smaller vessels is destroyed, and the latter become distended. With respect to the opinion advanced by Dr. Thomson, that increased action may form a part of moderate or incipient inflammation, or in some varieties, as the acute, be present from beginning to end, Dr. Hastings replies, that such a state is only that temporary excitement of the capillaries which precedes their debility, and which is inseparable from inflammation. "Certain stimuli," says he, "applied to living parts, produce an increased velocity of the blood's motion, and a contraction of the vessels. During this state of excitement, the part affected is so far from having any thing like the appearance of inflammation, that the size of the vessels is diminished and the part paler. But if the stimulus be long continued or increased in power, the small vessels which in the natural state admit only of one series of globules, become so dilated as to allow an accumulation of a much less fluid and redder blood in them, which loses its globular appearance, and moves much more slowly than that which previously passed through the vessels. The part now appears inflamed. If the stimulus be removed, the blood vessels do not soon regain their original state; time is necessary to allow them to recover their contractile power, so as to prevent the impetus with which the blood is propelled by the heart and larger arteries from keeping up the

dilated state of the capillaries." According to the same authority, if the stimulus which produces the inflammation be of a very acrid nature, debility of the vessels is frequently induced without any previous excitement; the blood in all the branches becomes very red, circulates very slowly, and in some vessels stagnates. Dr. Hastings rightly observed, that there was a temporary excitement of the capillaries inseparable from the act of inflammation, and he erred in not allowing this excitement, as he is pleased to call it, its due weight in the process. Like others who have adopted the theory of diminished action, he has overlooked the important phenomena which generally precede stagnation; and has attributed the whole chain of morbid effects to that debility of the capillaries which is seen in a later stage. In short, it has been too readily assumed, that the extreme vessels must be at once in a relaxed and enfeebled condition; while no allowance has been made for the share they at first take in the increased circulation.

The hypothesis of diminished action and slower motion of the blood in the capillaries of an inflamed part has been the most generally received, because its phenomena may be readily demonstrated with the microscope; while that of increased action of the vessels with accelerated circulation has been rather inferred from attendant circumstances. In the anxiety to support a favourite notion, or from some of the most material parts of the process having escaped attention, the series of changes incidental to inflammation have not been explained in connection, but separated and given only in part by the upholders of each doctrine. "Inflammation," according to the learned professor already quoted, "like every other phenomenon occurring in animated bodies, is to be regarded not as a simple event, but as one uniformly arising from the combined and complicated operation of powers, some of which are known, others at present unknown, to us. Simple views, whether of health or disease, however ingenious, can seldom be just. They have their origin in the spirit of system, not in the careful study and faithful enumeration of the complicated circumstances which concur in the production of all vital phenomena."

There is another error which has been committed by experimenters on this subject—namely, that of having confined their attention exclusively to the state of the vessels and the influence of stimuli over them, without having considered the manner in which these stimuli act. They have overlooked entirely the sensibility of the part on which they have been operating, and which modifies so materially the effects that follow. On this point, however, we confess ourselves unprepared to enter. We are unable to trace the influence which the nervous system has over the vascular, or rather the reciprocal relation existing between them; and, therefore, we cannot give a precise answer to the question—how far the intervention of nerves is necessary for the production of inflammation? The application of stimulants would lead us to think that the earliest change produced is in the sensibility of the part—a change

on which all the subsequent phenomena seem to depend, and without which, it is probable, they would never take place. In confirmation of this, we observe the rapidity with which inflammation advances in structures highly organised, compared with others where the organisation is less perfect; and it is only by reference to the sensibility of a part, and its connection with the nervous system, that we can explain the constitutional effects of local inflammation, and the great diversity we meet with in this respect. How far we may thus be enabled to account for the disease when proceeding from sources which we cannot trace and hence said to be spontaneous, we do not pretend to determine—it is possible that the order of occurrence is the same from an external as from an internal cause; and the fact, that in neither case does the morbid action yield till the sensibility of the part has first been lowered, is a strong proof of what we have stated.

Let us now briefly glance at the phenomena of inflammation. When mechanical violence, irritation, or stimulants are applied to a part, the first change induced is an alteration of its sensibility denoted by the occurrence of pain. This, without doubt the preliminary step in the process, is speedily followed by a change in the circulation; the capillaries are either constricted and the part becomes pale, or there is an increase in the quantity of blood and the part appears red. The difference observed at first in the colour and in the circulation, depends on some variety in the exciting cause. Thus, mechanical violence, irritation, and heat, have the effect simply of increasing the circulation; weak ammonia, or a moderate degree of cold, constricts the vessels and produces momentary paleness; while strong alcohol accelerates the motion of the blood, and then, in some cases, from the cold attending its evaporation causes its coagulation. It is impossible to divide acute inflammation into regular stages, as Kaltenbrunner has attempted; we believe it better for all practical purposes to consider the disease as consisting in numerous changes progressively going forward, the most conspicuous of which can only be described. Provided the source of local excitement is removed, the immediate effects may cease—the contraction and paleness of the vessels subside—the circulation becomes tranquil—and the part assumes its natural appearance. Whatever visible difference exists among local agents on their first application to the capillary vessels, we observe that their effects soon merge into each other by such insensible degrees as to make the ultimate results nearly alike. The interval of time which elapses between the immediate changes, and those which can be said to constitute inflammation, is various; in some cases scarcely a few seconds elapse, while in others, several hours will intervene. In the majority of instances this depends upon the severity and steady continuance of the exciting cause, partly also on the sensibility of the part affected, its importance in the scale of vitality, and the strength of the constitution; for, in repeating the same experiments on animals of the same class, whether warm or

cold-blooded, we have seen that inflammation is set up with very different degrees of celerity.

Whatever may have been the immediate effect of the stimulus applied, we next observe that the capillaries have their diameter enlarged; the part, from this circumstance, appears redder and slightly swollen—there is a distinct rise of temperature indicated by the thermometer, and the activity of the circulation is much augmented. The process of inflammation has now commenced. The irritation quickly extends to the larger arteries leading to the inflamed spot, and we can easily see with the naked eye that they are acting with increased energy. The balance among the different vessels is as yet maintained—the blood is circulating briskly—there are no signs of stagnation—the veins leading from the seat of disease are distended, and are carrying away the exact amount delivered into them by the arteries. It is quite an erroneous notion that the capillaries cannot be dilated so as to receive a greater quantity of blood, without supposing them, at the same time, to be paralysed or their contents stagnant. We have already remarked that, if they are endowed with a contractile power, dilatation, within certain limits, must be allowed to take place; and if ever any thing was made obvious to our senses, it is what we now contend for. Were the vessels constricted, we would not have a larger quantity of blood transmitted through them within a given time; for when this is brought on by the application of diluted ammonia, the part turns pale—the circulation is diminished—and, finally, comes to a stand when the constriction is complete. In the early dilated condition of the capillaries, we have the most convincing proof that there is no stagnation—the globules of blood are moving with increased rapidity—the veins leading from the inflamed spot are equally distended—and the appearance presented to the eye is very different from what is seen when the circulation is arrested. This, then, is properly the state of increased action; it is that which succeeds to the primary effects of the stimulus; it is the first stage of acute inflammation; the earliest sign of morbid vascular action.

The phenomena now detailed vary in intensity in different cases; no change, however, in the structure of the part is yet induced; no new products are formed; but the healthy functions of secretion and nutrition are suspended. It is during this early period that we find those changes commencing in the mass of blood, the study of which is of so much importance both in the investigation and treatment of inflammation. The violence and duration of this stage are not alike in all cases. In some, if the local cause is removed, the disease declines; in others, once the morbid action is set up, it continues to progress; and in both, the period during which the inflammation remains bears always a direct ratio to the violence with which it was established. It is better marked and more vehement in a warm than in a cold-blooded animal, as in the mesentery of the rabbit compared with the web of the frog's foot—a difference to be explained by the greater susceptibility and higher

organisation of the parts in the one over those of the other. This stage of acute inflammation has of late received the name of active congestion, in order to distinguish it from the changes which afterwards ensue. Those by whom it has been thus named have described the circulation as either "much quickened, or slow and embarrassed," and have attempted to separate it from what they are pleased to consider as real inflammation. But what name, we may ask, could they have chosen worse fitted to express their meaning, than one which is made to comprehend two directly opposite states? Why have they omitted to confer, on the last stage where the circulation is arrested, the absurd title of passive congestion? for in either case the part is full of blood. We should then have no more occasion to use the vulgar word inflammation; we should only have to speak of congestions. But if the changes we have described—and which are admitted by them to have taken place in the part—be not characteristic of genuine inflammation, by what signs are we to recognise it? Have we yet to learn that redness, pain, heat, and swelling, do not belong to inflammation, but are a part of active congestion; or, are we to search for the symptoms of the former in the stagnation of the blood and the formation of new products? Is not inflammation often a state of disordered vascular action, subsiding without leaving a single morbid trace behind it? or, are we now to give to such a case the more fashionable but foolish title of an *active congestion?* How much we have to lament that new terms should be employed to designate different stages merely of the same morbid action.

But let us return to our subject. The duration of the stage of increased action depends upon the violence with which it is set up, and the strength of the part in which it resides. Kaltenbrunner has satisfactorily shown that in tissues highly organised, and in which the circulation is rapid, as the mesentery of the rabbit, inflammation is not so speedily developed as in organs, like the liver, where the motion of the blood is slower; but that once it is established, it acquires a much greater intensity in the former than in the latter. At the commencement, and for some time after inflammation has begun, the circulation is equally supported by the capillary vessels, the blood is moving more rapidly, and double or triple the quantity seems to be transmitted through the part. Should every source of local irritation be removed, we find, in experimenting on the lower animals, that the morbid action may cease. The resolution takes place in various ways. In some, the action of the larger arteries which lead to the seat of disease declines, the redness disappears, and along with it the pain, heat, and swelling; in others, the vessels relieve themselves by the exhalation of a limpid or sanguineous fluid; or, if a mucous membrane has been affected, by an increase in its natural secretion, and not unfrequently by a discharge of blood. In some instances we have succeeded in putting an end to the disorder, by compressing the vessels leading to the inflamed spot, and keeping up the compression till the effects of

the local irritation have subsided; in others, we have procured resolution by keeping the part immersed in cold water, which causes the vessels to contract, and restores the original paleness.

In the majority of instances, in a warm-blooded animal, the local symptoms become more intense; from the sympathy called forth, the *vis a tergo* increases, there is a gradual subversion of the phenomena present, and a new and different series of morbid changes arise. Every thing conspires to exhaust the vital contractility residing in the delicate capillaries, and to paralyse their efforts to carry on the circulation. We will afterwards treat of the changes induced in the blood by inflammation—suffice it at present to remark, that it loses much of its fluidity, and becomes more viscid; that, in the minute vessels, its globular structure is broken down, and it appears like a red clotted mass, having a serous fluid effused around it. The increasing viscidity then of the blood, and the more vehement action of the larger arteries behind, tend, in particular, to destroy the tonicity of the extreme branches, which become still farther dilated, and lose their control over the fluid within them. The loss of power, unless when the local excitement has been unusually severe, comes on gradually, and in different situations; and it is easy for the eye to perceive the struggle made to maintain the circulation. In many of the extreme vessels the blood is now at rest, and the points of stagnation may be distinguished by their bright red colour. These phenomena are most conspicuous in the centre of the inflamed spot, for the circulation is still going on about the circumference.

The stagnation of blood, and the formation of cylindrical clots within the vessels—although at first confined to the capillary branches—soon extend to the arteries in immediate communication with them. As the inflammatory action increases, the redness assumes more of a livid appearance—the points of stagnation become more numerous, the effusion of serum is greater, lymph is poured out into the cellular interstices, and some of the over-distended vessels burst and allow their contents to be extravasated. Gendrin states that a section of an inflamed part exhibits, in some cases, towards the centre, an infiltration of pure blood surrounded with a gelatinous fluid; in others, a yellowish fluid resembling fibrin, and apparently made up of globules; farther from the centre of the inflamed spot, a reddish fluid containing a very few globules is found; while about the circumference there is only œdema. When the part is observed changing from a bright into a livid red, and the surcharged vessels are bursting, the inflammation is then at its greatest height; the healthy functions of secretion and nutrition were long before suspended, depositions of lymph and serum are forming in all directions, and a morbid change of tissue has now commenced. Kaltenbrunner remarks that he has observed, in the mesentery of the rabbit, globules of blood bursting through the sides of a vessel, and forcing a passage for themselves through the cellular tissue into another vessel; and that new canals are thus

formed by which the circulation is kept up. The bursting of the vessels, and the consequent extravasation of blood, are seen in every case where the local action is severe; but we doubt the formation of new channels for the circulation, by globules of blood forcing their way through the cellular interstices into other vessels, and are rather disposed to look upon it as a microscopical error. The rupture of the vessels has been attributed to disease of their coats, induced by the inflammation present; but from the rapidity with which this, in some cases, may be effected, time is scarcely afforded for an ulcerative action to extend through their tissue, and the laceration seems to arise simply from the pressure from behind. Some idea may be formed of the obstruction in the capillaries when inflammation is at its full height, from the circumstance that pressure with the finger will not then empty them of their blood. In a few instances, however, we have found that the coats of the larger arteries, near the seat of disease, had lost their elasticity, and were thickened from infiltration. Should the inflammatory action be rendered still more intense, and the part made to lose its vitality, the only other changes observed are, the colour passing from livid red into black, the blood still farther decomposed, coagula filling up the canals of the arteries in the vicinity, together with a great decline of the vital powers, evinced by the enfeebled state of the minute vessels, and the diminished force of the general circulation.

When treating of the immediate effects arising from a local stimulus, and of the first stage of inflammation, we remarked—that these frequently subsided, provided every source of irritation was removed; but when the morbid action has gone farther, we observe that little tendency is shown to abate till the congested vessels relieve themselves by the effusion either of serum or lymph, or by bursting and allowing their contents to escape. The transition from the state of increased action to that of diminished motion, and ultimately stagnation of the blood, generally takes place slowly, and occupies some length of time; but when the local irritation is great, the one may succeed the other so quickly that scarcely an appreciable interval of time comes between them. After the inflammatory action has subsided, the part always remains weak, and is so predisposed to a renewal of the attack, that a much less stimulus than was at first employed will suffice to rekindle it.

In viewing the leading changes going forward in parts labouring under inflammation, it would be possible to enumerate, but certainly not accurately to define, several intermediate stages; for we observe increased action and stagnation co-existing in different parts of the inflamed texture, and oppression and languor of the capillaries a considerable time previous to the circulation being entirely arrested. We may farther briefly notice, that the colour which the inflamed part takes on is modified by the agents employed to excite the disease. Thus, muriate of mercury gives to the vessels a brown tinge; diluted ammonia, or alcohol, produces a scarlet red; a saturated solution of common salt renders the part opaque, or, if employed

weaker, it imparts a purple hue. Some stimuli again are capable of removing that stage of inflammation where the vessels are relaxed and the circulation is impeded; others induce and increase it. Cold, nitrate of silver, and oil of turpentine, afford examples of the former; while muriate of soda causes instant dilatation of the capillaries, and augments still farther their debility. The effect of an agent in removing the inflammation is often temporary—the disease ceasing for a short interval, and again returning with renewed vigour; moreover, whenever a complete cessation is not obtained, we almost invariably find that the morbid action is much increased, and the part runs rapidly into a state of gangrene.

We mean not to recur to the question, what is the condition of the blood vessels in inflammation? If the phenomena, now so imperfectly related, be correct, they will furnish a sufficient answer to all such queries; and it must strike every one, as it has done us, that if the views entertained by the contending parties had been amalgamated and reduced to a proper order, we should have had ere now a satisfactory termination put to this discussion. It is much to be regreted that some portion of the valuable time wasted on this subject has not been given to matters of greater importance. However desirable it may be to have a precise acquaintance with the nature of inflammatory action, what is it, after all, compared to the diversified effects produced by this disease in the system of many of which we are almost completely ignorant? Theories are truly the soap-bubbles with which the grown-up children of science amuse themselves; while the idle crowd dignify these learned vagaries with the name of wisdom.

SECTION III.

LOCAL SYMPTOMS OF INFLAMMATION.

According to the artificial distinction drawn between Medicine and Surgery, our attention should be confined to inflammation occuring only in an external part; but the general remarks we mean now to offer will be found applicable to the disease wherever it may chance to have its seat. The symptoms of inflammation have long been divided by pathologists into two orders: those arising in the part affected, hence named the local—and those arising in the system in consequence of the local disturbance, named the general or constitutional. Each of these divisions will require our separate consideration, and it is important for the student to bear them in remembrance. We shall first treat of the local signs; and, under this head, discuss the changes observed during life, and those remaining after death.

Redness, pain, heat, and swelling, occurring together in one part of the body, denote the presence of inflammation. These four symptoms—to which some have added a sense of throbbing, and to which we would add disorder of function—were originally given by Celsus in his definition of inflammation; and, taken collectively, they furnish sufficient proofs of its existence. Inflammation, however, is a morbid action which extends to every organised tissue, causing such various results, and admitting of so many modifications, according to the structure affected, that although the symptoms now enumerated serve to describe it in a general way, they are by no means to be found in every individual case. Considered separately, indeed, there is probably not one them which can be said to indicate the disease, for they may all proceed from opposite pathological states; but viewed in combination, whether the whole are present or not, they leave us no room to doubt respecting inflammation.

The phenomenon which chiefly attracts our attention, when inflammation is external, is redness of the affected part. In order to be considered morbid, the redness must not only be permanent, but combined with some other symptom, such as pain. Redness, considered in this way, is the most constant, if not a pathognomonic, local sign of inflammation; one which can hardly ever be absent while the disease is present, and which seems essential to its constitution. In internal inflammation, the redness of course cannot be seen during life; but we scarcely conceive that where a part is inflamed it can fail to be red. There are some structures in the body, the vessels of which are so minute as not immediately to admit of red blood during inflammation; and much has of late been written, especially by some of the French pathologists, to prove that redness does not always accompany inflammatory action, and that a part may be seriously inflamed while it remains colourless. This, we are told, holds true with respect to certain transparent tissues, as the arachnoid of the brain, the cornea lucida, or the membrane of the vitreous humour. The first effect of inflammation on these is to produce opacity, which therefore becomes a sign of the disease; and, as a general rule, we may state, that parts which lose their transparency have their texture thickened and altered before they are injected with vessels carrying red blood.

But the doctrine, that redness does not invariably accompany inflammatory action, has not been fairly represented. In many cases, where no doubt is left us of inflammation having been the cause of death, it often happens that no traces of it are afterwards to be found. Properly speaking, inflammation is a state of disordered vascular action, which may subside without leaving any morbid effects behind it; and it is no argument, therefore, that the arachnoid, or other delicate membranes, have not been reddened by inflammation, because this is not to be perceived after dissolution. When a person is in *articulo mortis*, a strong contraction

of the blood-vessels takes place, by which the arteries empty themselves into the veins, and on which account the redness of an inflamed part often disappears. Sometimes the redness remains for a few hours and then subsides. We have seen the body of a child dying in an early stage of variola, uniformly red, and in a few hours turning pale and white. It is a remarkable fact, also, that in some cases of purulent ophthalmia, where the vision has been impaired, the inflammation will cease, and the transparency of the cornea be restored, immediately preceding death, from some other source. Before a part will retain its red colour, it is necessary that the inflammation shall have lasted such a length of time, and been so acute, as to cause permanent enlargement of the vessels, and stagnation of the fluid within them. This is demonstrated to us in our experiments on animals. In delicate transparent membranes, it may likewise be remarked, that inflammation always commences in the adjoining cellular structure, which is rendered vascular by the injection of its minute vessels. Where opacity, therefore, may be the earliest sign of inflammation, redness is still present in some adjoining tissue, and from this tissue the disease spreads to the proper membrane, which subsequently becomes thickened and vascular. We therefore conclude, that redness during life forms a constant and essential part of local inflammation.

The redness is always greatest towards the centre of the inflamed spot, because, as we stated in treating of its phenomena, the disease is most intense in that situation; from this it spreads and diffuses itself till it becomes lost in the affected tissue. The termination of the redness is in general indistinct, and no line of demarcation can be traced between the inflamed and the healthy parts; at other times it is more abrupt, and we may observe distinctly where the disease ends. The colour varies from a scarlet to a purple tint; and, as the part approaches to gangrene, gradually changes into black; a livid tinge is a sign of greater intensity than a bright red, and is always seen to increase with the severity of the local disorder. The depth of colour depends principally on the vascularity of the texture affected; it is greatest where the organisation is highest, and for the obvious reason that the morbid action is usually more intense.

The redness of inflammation is owing to an increased quantity of blood being transmitted to the part, and particularly to the distension of the colourless capillaries. If we look at the membrane of the conjunctiva, in the natural state, we observe that it is pale and transparent; if we examine the same structure under inflammation, we find that it is minutely injected with red vessels. In every instance where we have an opportunity of watching the local phenomena of inflammation, it is demonstrated to us, that not only those vessels which naturally carry red blood have their diameters sensibly enlarged, but that the otherwise invisible branches take on a similar mode of action. Both sets of capil-

laries are, therefore, dilated; and the distinction into the coloured and colourless artery, the *arteria non rubra* of Haller, is lost amidst the derangement of function created by inflammation.

When an incision is made into an inflamed tissue, a more than ordinary flow of blood ensues; and when the part is examined, it appears more vascular, its vessels turgid and increased in number. To account for the increase of vascularity, it was a favourite notion with some, that new vessels are developed either at the commencement or during an early stage of the disorder. The difficulty of accounting for such new formations, in cases where inflammation is suddenly induced, renders the hypothesis untenable. If we irritate the skin mechanically, or with a rubefacient substance, dip the finger in boiling water, or allow a foreign body to remain within the eyelid, redness, pain, and heat, immediately ensue, and the parts will go through all the changes consequent on an attack of inflammation. It is more correct in recent cases to ascribe the increased vascularity to the injection of the colourless arteries with red blood; and this is proved, *first*, by the fact of transparent parts, as the conjunctiva, becoming instantly red under inflammation; and, *secondly*, by the experiment originally made by Mr. Hunter. This individual froze and then thawed the ear of a rabbit; by these means he brought on inflammation. He next killed the animal and injected both ears. On dissection, the inflamed ear was found to contain a great number of vessels filled with the colouring matter of the injection; the uninflamed exhibited comparatively few. This experiment, confirmed since by repeated trials, proves the greater vascularity of an inflamed part, and points out, at the same time, the manner in which that vascularity is accomplished where the disease is suddenly induced. When inflammatory action, however, has continued long, we have reason to believe that new vessels are developed; but of these and their mode of formation we shall have occasion hereafter to speak.

Mere redness, in consequence of an increased quantity of blood being sent to a part, does not alone make up the act of inflammation. In blushing, the cheeks are rendered florid from the determination of blood towards them; yet we know that, on the cessation of the mental emotion, the redness will quickly disappear. Many similar examples might be given. It has been attempted to express the redness of inflammation by saying that it was preternatural; but we do not find that even preternatural redness constitutes inflammation. The changes induced in the skin by the exanthematous and other diseases afford the best illustration of the difference between preternatural redness and inflammation. In scarlet fever or measles, the skin over the whole body may be preternaturally red, yet it would be absurd to insist that it was inflamed; for inflammation taking place over such an extensive surface would quickly extinguish life; besides, the better marked and more vivid the eruption is, the constitutional effects are in general less severe. We make use of the phrase, increased vascular action or preterna-

tural redness, to express the condition of the skin in these cases, although we often apply the same terms when speaking of the condition of an inflamed part. In some instances, as small pox, it would appear that real inflammation is present, for the skin goes through some of the changes incidental to that disease, and forms purulent matter; besides, here, the more extensive the affection is —constituting the principal difference between mild and confluent small-pox—the greater is the danger to be apprehended. Let us compare, however, a patient having measles or scarlet fever with one labouring under erysipelas. In the last, we have real inflammatory action of the skin and adjacent tissue, and much pain is experienced when the surface is pressed upon; the system suffers severely althongh the affection should be limited; and, in short, the termination of the disease, as well as the symptoms present, leave us no room to doubt that the cutaneous texture is very differently situated from what we find it in either of the others. We may, therefore, have increased vascularity of a part so as to constitute preternatural redness, without inflammation being present.

Again, we observe, from experiments on animals and from some other circumstances occurring in the human body, that increased vascular action is often the first step towards the establishment of inflammation, and that a renewal or continuance of the exciting cause will convert the one state into the other. If we apply friction to the skin, we simply cause increased redness; but, if the friction is too long continued, we bring on inflammation. Should a foreign body lodge in the conjunctiva, redness and turgescence of the vessels ensue; if the extraneous substance is withdrawn, the increased vascular action subsides—if allowed to remain, inflammation follows. Mr. Hunter long since accurately observed, that such is the way in which inflammation generally commences; and it would appear that we are often at a loss to distinguish between that kind of vascular action which occurs in health, and that which leads to the formation of disease. On the one hand, we find it existing in a preternatural degree, and for a length of time, without producing any bad effects; while, on the other, the operation of the same cause speedily brings on disordered action. Hence redness, as a part of inflammation, must not only be permanent before it can be considered morbid, but must be accompanied by some of the other local symptoms of the disease.

Lastly, more blood may be accumulated in a part, as in what is termed *congestion*, without inflammation being present. We have no wish to enter into any controversy on this subject; its importance and the frequency of its occurrence have been grossly exaggerated; it seems to be ill understood, and, consequently, much absurd reasoning has been adduced respecting it; and it has given rise to great confusion in practical medicine. In the most common acceptation of the word congestion, we understand the blood to be collected in the venous trunks, or in those branches of the capillaries which form the incipient veins, and in this sense only we can

comprehend its meaning; wherever the colourless arteries are distended with red blood under the circumstances we have mentioned, we are entitled to consider the part as inflamed. The phrase congestion, however, too fashionable or mysterious to be lost sight of, has been applied to other states where blood is accumulated; and coupled with the adjective active, has, as already mentioned, been improperly made to include the early stage of inflammation.

The next local symptom which we shall consider, is pain. When irritation is applied to a local part, the first change induced is in the function of the nerves, by which pain is created and which leads to increased vascular action and all the subsequent phenomena of inflammation. This is the way in which the disease seems to be established, and the nerves may so far be regarded as the safeguards of the constitution, warning us of the presence of local mischief, which might and does make dangerous inroads where the sensibility is impaired. It is by means of this symptom that we judge of the seat of internal inflammation; it is in general the occurrence of pain, and not of redness, which first awakens the patient to his condition; and although not an unequivocal, it is, nevertheless, one of our principal guides, by the presence or absence, or degree of which, we judge of the intensity of inflammatory action in parts concealed from view. In consequence of pain or uneasiness, we are made aware of the existence of organs which, in the healthy state, transmit no sensations; and hence pain is a valuable sign, at least, of derangement in the natural function of a part.

Pain is not, however, a symptom of inflammation only, for we can show that it may be excruciating while inflammation is absent. In various forms of neuralgia, the pain may be so violent as to make the sufferer cry aloud; yet where opportunities have occurred of examining the nerves after death, no marks of inflammation have been found, nor does the complaint yield to depleting remedies. The same thing is observed in many other nervous affections, and the mere existence of pain in a part is not to lead us to infer that it must necessarily be inflamed. Again, the most fatal inflammation may be going forward where pain is wanting; and the disease, attended with this peculiarity, is always of the most dangerous description. In many cases of typhus, especially in advanced life, the lungs may be extensively inflamed without any other symptom than cough or a hurried respiration; and it too frequently happens, from the patient being rendered incapable by the fever of expressing his proper feelings, that the local disease is overlooked. Inflammation of the substance of the brain is sometimes marked by the same peculiarity; and singular as the statement appears, this organ, though the source of feeling to other parts, seems itself possessed of little sensibility, and, unless in its diseases we had other symptoms to guide us than mere pain, we should often be misled. To point out how far inflammation may take place independent of pain, we may mention the case of paralysis of the extremities, where blisters may be applied or burns inflicted without the consciousness of the

individual. Moreover, pain does not invariably point out the exact seat of the disease. Thus, in inflammation of the liver, pain is felt at the top of the right shoulder and down the arm; in hip-joint affections, the chief uneasiness, at first, is at the knee; and hence a wrong reference is often made to the locality of the disorder.

The production of pain is chiefly regulated by the nature of the tissue in which the inflammation resides; and no anatomical knowledge would have enabled us to say *à priori* where increased sensibility would have been most exhibited. We should likely have concluded that, in textures where nerves are plentifully distributed, inflammation would have created the greatest pain; yet experience teaches us that parts possessing only a moderate share of organisation, and even devoid of sensibility in the normal state, become unusually painful when inflamed. It may be stated, as a general law, that, in all dense tissues or those least susceptible of swelling, the greatest amount of pain is experienced; and hence the reason why bones or cartilages take the lead of other structures more highly organised. There is thus often a wide difference between containing and contained parts. When the substance of the brain is the seat of the disorder, little or no complaint of pain is made; the individual for the most part is unconscious of his condition, and affected with stupor or typhomania; but should the inflammation be in the membranes, or should these subsequently participate, the pain becomes severe, accompanied with watchfulness and furious delirium. The same law, with respect to pain, holds true when the substance of the liver or its serous covering is affected; yet in all cases, the danger is proportionally greater, when inflammation is seated in the interior of an organ than in its investing membrane.

The mucous or cellular tissue, above all others, furnishes the least amount of pain during inflammation. Of the former, a very familiar example occurs in nasal catarrh. Here the lining membrane is inflamed and swollen, the person expresses his condition by saying that his head is stuffed, a thin discharge succeeds which terminates the disease, but all along we have no complaint of pain. In acute inflammation of the bronchial tubes, the morbid action is indicated more by the general febrile symptoms, the hurried respiration, the mucous râle heard over the chest, and the discharge of sputa, than by any uneasiness experienced by the patient, and, which seldom amounts to more than a sense of constriction. In inflammation, likewise, of the mucous membrane of the intestines, pain is but a secondary symptom; and, in either of the two last cases, the comparison is striking, when the serous covering is the seat of disorder. It is obvious that a mere reference to physical structure will never explain the diversities we meet with in this symptom, else parts possessing the fewest nervous filaments and having the smallest share of organisation should afford the least pain. In all lax structures, the natural sensibility is but slightly exalted; while in dense organised tissues, it may reach the highest pitch. The degree of pain varies from slight tenderness to the touch

up to the most acute suffering, and depends on the nature of the tissue affected, the intensity of the inflammation, and probably, also, the idiosyncracy of the individual. Next to the particular seat of the disease, the severity of the local action has undoubtedly the greatest influence in creating pain; but as no just estimate can be formed of the intensity of inflammation in any two cases, no accurate comparison can be drawn.

We need not discuss the question, What is pain; or in what does it consist? It appears to be made up of some mental change; and although originating in, or kept up by, local disease, yet it is solely dependent for its production on a healthy state of the brain and the continuity of the nerves proceeding therefrom. In proof of this, we may quote the absence of pain when the function of the brain is disturbed; or the paralysis of the extremities, where inflammation may be induced without the consciousness being awakened. We may farther observe, respecting the pain of inflammation, that it seems to consist in an exaltation of the common sensibility diffused over the body; while that which is peculiar to parts, as rendering them instruments of sense, is lost or impaired. During the prevalence of catarrh, the sensibility of the lining membrane of the nose is so great that the minutest particles floating in the air excite sneezing; but the sense of smell is for a time suspended. And the same thing may be observed of other organs.

It is important to learn that pain, or a painful condition of the filaments of nerves, arises from more sources than one; hence pathologists have laid down rules for distinguishing spurious or spasmodic from inflammatory pain. The word spasmodic is ill chosen, as conveying an idea of muscular contraction; for although pain may arise from the violent spasmodic action of muscles, as in tetanus, it more frequently exists as a peculiar affection of a nerve, as in neuralgia. When employed to denote that pain, arising from other causes than inflammation, occurs in paroxysms or spasms, the phrase is more appropriate, for this is one of the principal marks of distinction. Pain, resulting from local inflammation, may be described as constant, though not uniform in degree; that arising from other sources and commonly called spasmodic, is attended with remarked remissions, and not unfrequently there is a complete intermission between the paroxysms. Constant tenderness to the touch, therefore, with nearly a steady degree of pain, enables us to distinguish inflammatory from spurious cases, where patients suffer from marked exacerbations, lasting for a short period, and then perhaps completely disappearing. It may be laid down as a general rule, that inflammatory pain is aggravated by pressure, or motion, or whatever tends to disturb the actions of the affected part, while spasmodic pain, on the contrary, is generally relieved. The most prominent example of this is afforded in enteritis and colic, which may readily be distinguished, independent of constitutional symptoms, by the application of pressure alone. We can easily explain why pressure should aggravate the pain of inflammation, and why

it should relieve that arising from colic; but there are many cases of spurious pain, where pressure operates in a way of which we know nothing, while there are others in which it produces no effect whatever.

There has been apparent exception made to the law now laid down, and which, coming from high authorities, deserves consideration. It has been stated that, when an inflamed part is wholly and gradually compressed, the pain will be relieved; and that it is only because the pressure is partial the pain is aggravated. To ascertain, therefore, that pain arises from inflammation, we should compress the part suddenly and partially, as gradual and equable pressure over the whole affords relief. Our success, it is said, in such cases, depends on emptying the vessels of the inflamed structure completely of their blood; a circumstance which, unless a detached part, as a toe or a finger, was the seat of disease, we believe to be perfectly impossible. Founded, however, on this principle, Velpeau and others have suggested compression as a means of cure in erysipelas; but there are substantial reasons for declining the recommendation. We must confess that we have never yet had the misfortune of meeting with a case, where the mode merely of applying pressure rendered us in any doubt as to its exact nature; and we look on the whole as an ingenious quibble, more fitted to amuse than instruct.

The kind of pain experienced in inflammation varies according to the seat and intensity of the disease; but it is very different at all times from that which occurs in spasmodic cases. Sometimes it is acute, at other times dull; sometimes it is of a lancinating or burning nature, or of an itchy or prickling kind; frequently it is a throbbing pain, increased at each pulsation of the vessels; and, in some of these cases, as whitlow or cephalitis, aggravated, by a dependent posture. Spasmodic pain, again, is often violent, lasting a few seconds and then remitting; in neuralgia, it more resembles electrical shocks than anything else to which it can be compared; occasionally, it is of a stinging description; but it never consists of the mixture of pain and heat—the true burning pain of local inflammation. In colic, the pain is described, in some instances, as being of a cold nature, and certainly it is very different from what is experienced in genuine enteritis. Expression of countenance is not of great value as a sign of pain from inflammation, and is only observed in cases which have lasted a few days; the invasion of acute disease may be so rapid as to admit of little change being effected on the features.

The signs of distinction now given, enable us, on all occasions, to separate inflammatory from spasmodic pain; and, were anything farther wanted to complete the diagnosis, it would be found in the amount of febrile disturbance in the system, and in the origin and progress of the two cases. It is principally in delicate females, the subjects of hysteria, that we meet with cases similating inflammation; in whom these affections often spring from some mental

sympathy, and for the relief of which we have often known the most vigorous depletion imprudently put in force. We ought to recollect, however, that spasm may induce, or be followed by, inflammation; and thus these two states may be blended together. In fine, sudden remission of severe pain, without sufficient cause, should put us on our guard, either that the case is not one of inflammation; or, if so, that some change of action has arisen.

Lastly, how are we to account for pain as a local symptom of inflammation? The hypothesis has been started, that new nerves are developed; but pain is one of the earliest effects of a stimulus, and there is not time allowed for the new formation. Again, it has been supposed that the extremities of the nerves themselves were inflamed; but we have no proof of this on examination after death; besides, were it true, the more copiously then a part was supplied with nerves, the greater should be the amount of pain. In all dense structures, as bones or fibrous membranes, where swelling is most resisted, the pain, as we have said, is great compared to that arising in parts which are lax and readily admit of distension. The mechanical compression, which the minute branches of the nerves must suffer during inflammation, explains more satisfactorily the origin of pain than any other hypothesis that has been brought forward. The ramifications of nerves accompany those of arteries to their invisible terminations, and are enclosed with them in the same sheath; hence the over-distension of the coats of the vessels and the pressure from newly effused fluids, must cause undue compression of the nervous twigs, and give rise to unnatural sensations. If, therefore, the pain in the first place proceeds from over-distension of the coats of the blood-vessels, and, secondly, is increased by the effusion of fluids into the part, it follows, that the denser the structure, the greater must be the pressure exerted on the extremities of the nerves. In this way only can we account for the pain accompanying inflammation in tissues having but a moderate share of organisation, and displaying little or no sensibility in the healthy state. And, in farther corroboration of this view of the local cause, we may remark that, in certain inflammations, the pain is much increased during the distension of the arteries at each contraction of the ventricle; and, on which account, it has sometimes received the name of pulsatile.

Heat, as a symptom of inflammation, is not in reality so great as we might often be led to expect from the feelings of the patient. Mr. Hunter made numerous experiments in order to determine whether or not any increase of temperature takes place. Having brought on inflammation in the chest, abdomen, rectum, and vagina of animals, he then introduced the thermometer to ascertain the degree of heat, and he came to the conclusion, that it seldom exceeded that of the blood as it issues from the left ventricle of the heart. In parts at a distance from the centre of the circulation, the temperature is always lower; but Mr. Hunter believed that in them, it never reached the natural standard during inflam-

mation. He operated on a man in St. George's Hospital, for hydrocele, with a view to peform the radical cure. The tunica vaginalis being opened, he immediately introduced the ball of the thermometer by the side of the testicle; the mercury rose exactly to 92°. The cavity was filled with lint, dipped in salve, (an old method of radically curing hydrocele,) in order that it might be taken out at will. The next day, when inflammation had come on, the dressings were removed, the ball of the thermometer was introduced as before, and the mercury now rose to 98¾°; but even this was probably not equal to the heat of the blood, at the source of the circulation in the same man. This experiment, it is added, was repeated more than once, and with nearly the same results.

In consequence of such high authority, many have adopted the opinion that there is no rise of temperature in inflammation; but whatever value we attach to Mr. Hunter's experiments, we cannot, in the present day, coincide with the inferences which he drew from them. We believe that, were it possible to measure the degree of heat, it would be found in almost every instance to be increased. It is quite unreasonable, for example, to argue that there is no increased heat, because the temperature of an inflamed part may not exceed that of the blood at the source of the circulation; for if it rises two or three degrees above that which is common to the part, then we contend that the heat is preternatural. The rise of temperature is best observed in the extremities, as they are at the greatest distance from the heart's action; but the same thing would be found, though in a less degree, in parts in the more immediate vicinity of the chest. The standard heat of the blood may be reckoned about 98°, varying from 96° to 100°, while that of the inferior extremities is usually six or eight degress below. When, accordingly, inflammation takes place in the latter, the heat may scarcely average that in the chest or axilla, yet the natural temperature of the parts is elevated several degrees; and it is from this circumstance being overlooked, that the opinion has gone abroad, of the heat not being augmented during inflammation.

From the warmth experienced when the hand is laid over an inflamed part, and from the great complaint made of this symptom in some cases, we might be led to expect that the rise of temperature would be very considerable. It is necessary to be aware, however, how liable we are to be deceived by the hand, in judging of heat or cold. If the hand is cold when applied, the inflamed part will indicate a degree of heat not in reality present; if warm, the temperature may be considerably elevated without our being able to detect it. It is better, therefore, not to trust to the hand, but to use a thermometer wherever it is practicable. We have now so many convincing proofs of the temperature of the body being raised above the natural standard, that no arguments to the contrary can invalidate them. In common fever, the mercury in the thermometer placed under the tongue or in the arm-pit will rise to 107°, nine degrees above the heat of the blood in the aorta, supposing it to

average 98°. In scarlet fever, we have found the heat ranging from 100° to 112°. It has been stated, by accoucheurs, that the uterus, during labour, will cause the thermometer to rise as high as 120°; and we observe in some spasmodic diseases, as tetanus, that the mercury will reach 106° and 107°. In erysipelas of the lower extremities, we have found the heat as high as 107°, and in one case 108°; and it is not correct, as Mr. Hunter supposed, that, in parts at a distance from the heart's action, the temperature never exceeds that of the blood at the source of the circulation. It is only where inflammation is external that the degree of heat can, of course, be measured, although we have little doubt that it would be found increased in most other cases; and we must again insist on our position that, wherever the temperature rises above that which is common to a part in health, whether that rise is greater or less than the natural standard, then preternatural heat is unquestionably indicated.

We do not consider increased heat an unequivocal sign of inflammation. Parts may be unusually warm where no morbid action of this kind prevails; and, again, a sense of preternatural heat is frequently not complained of, where intense inflammation is going on. It is principally when inflammation affects the extremities, that heat becomes a prominent symptom, as in these the circulation is more languid and the temperature always lower. In pleurisy, for example, pain is the most remarkable local symptom; while, in whitlow or erysipelas of the leg and foot, both pain and heat predominate. There are, however, a few cases of internal inflammation, where increased heat is present. In enteritis, preternatural warmth is often experienced by the patient; and in phrenitis, it is perceptible when the hand is placed on the naked scalp. Heat, therefore, is a valuable means of detecting inflammation of the brain in children, or in those who may be incapable of expressing their proper feelings.

We can offer no satisfactory explanation of heat as a symptom of inflammation, without reference to its production over the body; and of this, we may at once confess that we know almost nothing. It was first supposed by Dr. Black—and the theory was supported by Crawford and others—that the formation of carbonic acid in the lungs was attended with an evolution of heat similar to what we observe results from the union of carbon and oxygen in ordinary combustion. The heat was, however, rendered latent in consequence of the greater capacity of the arterial blood for caloric; but was again given out, during the transition of the red into venous blood, in the general circulation. Dr. Adair Crawford estimated the specific caloric of arterial blood to exceed that of venous by 14.5°, an amount which has since been reduced by Dr. Davy to 1°. Nevertheless, the capacity of red blood for caloric being greater, the latent heat was set free wherever the change into venous was going on.

Such was the generally received theory till the experiments of

Sir B. Brodie were made known, and which have gone to prove that the nervous system is concerned in the production of animal heat. After securing the vessels of the neck, Sir B. Brodie killed two rabbits by decapitation; in the one, artificial respiration was kept up—the other was left untouched. The circulation and respiration went on perfectly in the former, and yet it cooled more rapidly—the thermometer standing at 97° in the rectum of the one, and at 98° in that of the other. Sir Everard Home divided the nerves distributed to the growing antler; its temperature immediately fell several degress, but rose again in a few days higher than that of the opposite horn. The inference from these trials would be, that animal heat does not depend on the changes induced in the blood at the lungs.

The experiments of Sir B. Brodie have been repeated and contradicted by Legallois; and Drs. Hastings and Philip have subsequently shown, that the more rapid cooling of the first animal was owing to too large an inflation of the chest with the cold air, and that artificial respiration, moderately kept up, retards instead of accelerating this process. There are several circumstances which point out the changes taking place in the blood, as the principal source of animal heat. When the main artery of a limb, as the femoral, is surrounded with a tight ligature, the temperature occasionally falls so low that we are obliged to have recourse to artificial means to guard against it. The farther a part is removed from the source of supply, the natural temperature is proportionally diminished; hence that of the lower extremities is always less than of the upper. In cases where the blood is imperfectly arterialised, and a dark mixture is circulating through the vessels, as in some malformations of the heart, the natural heat of the surface is deficient; and the same thing may also be observed in asthma, when the respiration is not sufficiently performed. Dr. Wilson Philip applied the galvanic influence to the arterial blood of a rabbit immediately on its being drawn; an evolution of heat amounting to 2° or 3° took place, and in the meantime the blood acquired a modena hue. In venous blood, on the other hand, no rise of temperature could be effected. Dr. Davy has admitted that the specific caloric of arterial blood exceeds that of venous at least by 1°, and, it being apparent that this excess is discharged during the transition of the one into the other, it may be inferred that, as this change takes place in the capillaries, it is there where animal heat is first evolved.

Admitting, however, that the changes in the blood constitute a source of animal heat, it is still doubtful in what manner the specific caloric of the arterial fluid is acquired. Dr. Crawford supposed that the carbonic acid of respired air was formed in the lungs by the direct combination of the oxygen with the carbon of the blood, and that the caloric, necessarily evolved by the combination of these two elements, was absorbed, and became latent in the arterial fluid. But there are strong reasons for thinking, that the formation of the

carbonic acid does not take place at the lungs, but that it exists previously in the blood, and is merely thrown off at these organs. The theory, that the volume of oxygen which disappeared during respiration was greater than that of the carbonic acid exhaled, was at one time overturned by the experiments of Allen and Pepys, which went to show that the two were exactly equal. The more recent researches of Edwards and Dulong have proved, that the amount of oxygen consumed is greater than that of the carbonic acid given off; and, consequently, it has been inferred, that the whole of the oxygen may as well be absorbed into the blood as part going to the formation of the fixed air. Dr. Edwards has further proved that, in animals confined for various periods of time in pure hydrogen, nearly as much carbonic acid is exhaled as if they were breathing pure air, and much more than could, by any possibility, have existed in the lungs previous to immersion in the inflammable gas. It would appear, from other experiments, that similar results are obtained when animals are made to breathe pure azote; and, in the human subject also, the full quantity of carbonic acid has been thrown off, while the lungs were inflated with nitrogen.

These facts point out that the carbonic acid of expired air is not formed in the lungs, but merely extricated at these organs from the venous blood; and if this be true, then there would be no union of oxygen with carbon in the chest, and no caloric evolved to be absorbed by the arterial blood. In whatever way the changes on the circulating mass within the lungs are brought about, one thing is evident, that a volume of oxygen is consumed equal to, if not greater than, the carbonic acid expired; and whether this volume of oxygen combines in part with the carbon of the blood to form the fixed air, or passes wholly into the arterial fluid, in either case, from the condensation which ensues, a rise of temperature must follow.

Dr. Edwards has likewise shown that, in young animals, less oxygen is consumed in respiration, and that they have a less faculty of generating heat; moreover, he considers that this power is increased or diminished by the quantity of oxygen taken into the lungs. But, on the other hand, it may be observed that, if the power of generating animal heat depended solely on the consumption of oxygen at the lungs, then it might be expected that frequent inspirations would cause a great increase of temperature. No such effect, however, follows the attempts of the kind, and we find the only way of accomplishing this is to accelerate the circulation, and thereby indirectly create a necessity for fresh air. In a patient at St. George's Hospital, with injury of the cervical vertebræ, the heat rose to 11° at a time when the respirations did not exceed five or six in a minute—a proof that the development of heat is not in proportion to the consumption of oxygen.[1]

[1] Medical Gazette, June, 1836.

Dr. Wilson Philip admits that the action of the air upon the blood in the lungs is the principal source of animal heat, but considers its extrication throughout the system as a sort of secretion, in which the nervous system is directly concerned. This opinion derives support from his experiments, which show the effect of galvanism in causing an evolution of caloric and the transition of the arterial blood into venous. Dr. Edwards believes that the power of generating animal heat does not depend on any single function, but that it is intimately connected with respiration, circulation, and secretion; and, moreover, that it cannot undergo any sudden and permanent alteration in health. If we understood the influence which the nervous system has over the capillary circulation, we should be better able to explain the phenomena in question. There can be little doubt, however, that the process of secretion and nutrition are intimately concerned in the production of heat over the body, for whatever tends to disturb these, has the effect of altering, in some way or other, the animal temperature.

Chossat has shown that injuries of the nervous system, which lower the temperature, are identical with those which diminish the functions of secretion and nutrition. In Sir B. Brodie's experiments, these functions were necessarily suspended; and we can readily understand why the inflation of the lungs would then accelerate the cooling of an animal. It would appear, however, that there are some injuries to the nervous system, as those done to the spinal cord, where a remarkable evolution of heat takes place; and, in the case already recorded, the temperature reached as high as 111°. It is probable that a great difference will be observed, as respects animal heat, between cases where the functions of secretion and nutrition are completely arrested from the annihilation of the nervous influence, and those wherein they undergo some derangement in common with the vascular system. It is in this latter way that we would explain the temperature, both in fever and inflammation, being so many degrees above the natural standard; and by which we would reconcile also the statements of Dr. Hastings, who has seen the pulse in fever at 45, and the heat of skin at 105°; and, in hydrocephalus, the former at 60 or 70, while the latter was at 100°.

It has been argued,[1] in proof of the heat in inflammation being dependent on the blood, that it never exceeds the standard acquired by that fluid in the aorta; but we have already shown that Mr. Hunter himself was deceived on this point. In experimenting on animals, we observed that, as soon as inflammation is established, and every vital function in the part is deranged, the temperature then rises; but it appears to us impossible to explain this increase by the state of the circulation alone, without taking into account the changes induced in the nervous system, so far at least, as the latter is connected with the functions of secretion and nutrition.

[1] Thomson's Lectures on Inflammation.

The evidence on the production of animal heat is still conflicting; and, until we are able to account satisfactorily for this vital process in the state of health, no just explanation can be expected of its increase or diminution in disease.

Swelling, although usually enumerated among the local symptoms, ought rather to be regarded as an effect or product of inflammatory action. It is chiefly seen when inflammation occurs in the more external parts, and can seldom be discovered when the disease is internal, till on examination after death. Perhaps, the brain is the only tissue which will not admit of swelling; the contents of the cranium cannot, properly speaking, admit of increase; and when effusion of any kind takes place here, some of the vessels are unduly compressed, and we explain the symptoms which arise, by supposing a portion of the nervous matter to be deprived of its supply of blood. Lax tissues, as the cellular or mucous, are those in which swelling most readily occurs. Hence dense structures, as bones or fibrous membranes, do not become thickened till the morbid action has been some time present. Swelling has frequently the effect of relieving the inflamed vessels; at other times it aggravates the local symptoms, by creating a feeling of tension, as when the scalp is the seat of disorder.

Swelling, in the first place, is caused by the greater distension of the blood-vessels, and especially the colourless capillaries. The conjunctiva, for instance, from being smooth, becomes turgid and slightly elevated when inflamed, and the inequality of surface thus produced imparts a sensation of a grain of sand or other foreign body being within the eyelids. After inflammation has been a short time established, an effusion of fluid from the over-excited vessels takes place. The effusion consists either of serum tinged with the colouring matter of the blood, or a mixture of serum and coagulating lymph. When the local action is intense, some of the distended vessels burst, and blood is extravasated, so that the section of an inflamed part occasionally exhibits blood, lymph, and serum combined. Should the effused fluid consist principally of serum, the swelling is soft, and disappears quickly after the inflammation has ceased; when much lymph is thrown out, the part feels firm and tense, and the hardness remains for some length of time. In general, the effusion is made up both of serum and lymph, and the swelling is partly soft and partly hard, and disappears sooner in one situation than in another. The induration left after inflammatory action is often a source of great inconvenience, and, from the length of time required for the natural absorption of the lymph, causes much uneasiness in the mind of the patient. Swelling, in inflammation, may also be partly caused from the function of the absorbents being interrupted by the pressure from the newly deposited fluids. Swelling, from collection of blood or serum, must not be mistaken for that arising from inflammation. Thus we may have blood effused from a blow, or serum collected in the cellular membrane, as in œdema, without any morbid action being present;

but we know of no instance where lymph is poured out, unless as a product of inflammation. Swelling must, therefore, be combined with other symptoms before it can be received as any evidence of inflammatory action, and would, as we have already hinted, be more properly included among its effects.

The throbbing of an inflamed part arises from the obstruction the blood meets with in its passage through the dilated capillaries, as is illustrated by the experiment of tying a ligature round a finger. It is generally found where inflammation is limited in extent and violent in character, and is peculiar to some forms, as whitlow and cephalitis. It may be present from the commencement, and gradually decline as the disease abates. When occurring at a subsequent period, from the greater intensity of the inflammation, it often indicates that suppuration will ensue. The local pain is always increased at each pulsation, and has hence been denominated pulsatile; it is produced during the dilatation of the vessels. Where throbbing is met with, we commonly find that it is much increased by postures which favour the flow of blood to the part, and prevent its free return by the veins.

Another local symptom, and one which we hold in as high estimation as any yet treated of, is functional derangement of the part affected. There is not, nor can there be, any organ in the body which does not exhibit this when attacked by inflammation; hence its importance in ascertaining disease. The truth is self-evident, that a disordered organ can never perform its functions rightly—the coincidence therefore inseparable, of a disordered function implying a disordered organ. Let us give a practical illustration of the value of this symptom. In inflammation of the substance of the brain, neither redness or swelling are visible, there may be no preternatural heat indicated when the hand is laid on the scalp, and the patient may be rendered incapable of expressing his proper feelings. What other symptom, then, have we to point out the seat of the local mischief, save the derangement manifested in some of the natural functions of the affected organ? How often, in cases of typhus fever, should we be deceived as to the existence of fatal inflammation in the lungs, were it not for the hurried respiration, the accidental cough, and the phenomena indicated by the stethoscope? And in inflammation of the mucous membrane of the intestines, are not the discharges from the bowels the most conspicuous local signs of the disorder? Derangement of function at once proclaims the seat of disease; and there is no part which has an office to fill in the animal economy that can be exempt from this law. We are, of course, far from wishing to assert that functional disorder is attributable to no other source than inflammation; we wish only to state that, in combination with other evidence, it stands invaluable as a local symptom.

The alteration occasioned in the function of parts by inflammation is modified according to the violence and duration of the disease. The most common effect is to suppress or alter the secre-

tions, as when the kidney or liver is the seat of disorder; in other structures, as the mucous, the discharge is both vitiated and increased. Suspension of a natural secretion, however, is not an invariable mark of inflammation. In suppression of urine, when other symptoms are wanting, we should not be justified in adopting bold depleting measures. Functional disorder, as a result of inflammation, arises in two ways; either from the mere violence of the local action, which, as soon as subdued, leaves the part again fit for its office; or from morbid change of structure, where, although the inflammation may have ceased, the derangement of function remains behind. As a symptom of inflammation, therefore, we have to consider whether it is the immediate and transitory effect of disordered action, or whether it springs from some permanent alteration induced in the natural structure; for our prognosis with respect to its future disappearance, in the two cases, is very different. In whichever of these ways disorder of function is caused, we find that it is a never-failing accompaniment of inflammatory action, and therefore a useful guide to the disease when occupying an internal part.

Lastly, we shall briefly allude to the local signs remaining after death. We do not mean to detail, at present, the change of structure left by inflammation, but simply to enquire how far the symptoms now treated of correspond with the condition in which we find an inflamed part. This is a subject of great importance in a medico-legal point of view, and one in which serious mistakes are liable to be committed. In making *post mortem* examinations, nothing appears more surprising than that no traces of derangement can be seen in cases where we were certain, from the symptoms during life, inflammatory action had been going on; and again, that we should meet with evident marks of the disease where no suspicion of its existence had previously been entertained. When inflammation attacks an organ essential to life, as the heart, death may ensue before time has been allowed for any change of tissue to be effected. Strictly speaking, inflammation is a state of disordered action incompatible with that possessed by a part in health; and the tendency of which is, by long continuance only, to give rise to morbid products. It may so happen, therefore, that no apparent alteration is to be discovered, merely because the disease has proved fatal before these changes could be brought about.

Redness and swelling are the only local signs of recent inflammation which are to be found after death; the pain and heat, the sense of throbbing, and the disorder of function, must have necessarily ceased with life. Redness, however, is not always to be observed; neither are we to infer from its presence that inflammation had taken place. We are quite certain that the redness of an inflamed part may disappear after death, as we witness when the skin or conjunctiva has been effected; hence we are not to conclude, provided there was good evidence to the contrary during life, that a person may not have died of inflammation, simply because the part

does not afterwards appear red. In order that the redness be permanent, the inflammation must have reached that stage where the vessels are dilated and have lost their contractile power, and where the blood is stagnant and cannot be urged forward by pressure. In such a case, we have generally other signs of the disease, as effusion of serum or lymph. Should the inflammation terminate fatally, before these changes in the state of the minute vessels have taken place, then the redness disappears in consequence of the general contraction occurring throughout the whole of the arterial system at the moment of dissolution.

Neither is mere redness any proof of a part having been inflamed. Dr. John Davy[1] first showed, and his observations have since been confirmed by Andral, that the colouring matter of the blood may transude so as to impinge the neighbouring solids and redden their contents. Unless a body is examined within twenty-four hours after death, in warm climates where decomposition proceeds more rapidly, Dr. Davy considers it impossible to discriminate between the redness of inflammation and that caused by transudation. During life, the colouring matter of the blood does not even tinge the lining membrane of the vessels along which it moves; but after death, and especially when decomposition has commenced, the solids loose their cohesion, become soft and porous, and allow the blood to transude through their substance. The period at which the examination is made, is, therefore, a matter of great importance, in all cases.

The transudation of blood through the solids is much influenced by the manner in which death has happened. Thus, where it has arisen suddenly, as from lightning, drowning, or suspension, the tendency of the blood to transude is much increased. In some of these the contractility of the arteries is instantaneously destroyed, and these vessels, as well as the veins, remain full of blood; in all of them the greater fluidity of the blood allows it to ooze more readily through the tissues, and thus accelerate decomposition.

Position has also a remarkable effect in causing redness, by allowing the blood to gravitate towards the most depending parts. Thus, in bodies lying in the ordinary way, the most prominent points on which they rest, as the back part of the scalp, the shoulders, and nates, are reddened. The posterior portions of the lungs, for the same reason, usually contain a considerable quantity of blood; and were we to set this down as a sign of inflammation, we should be guilty of a gross error. Notwithstanding the influence of gravitation, there is a tendency to congestion in all internal organs, arising from the inability of the heart and arteries, during the last acts of life, to transmit the blood to the surface of the body. If a part which is naturally pale be placed in such a position as to favour the gravitation of the blood, and then examined at the end of three or four days it will be found to have assumed a bright red. Rigot

[1] Medico-Chirurgical Transactions, Vol. X.

and Trousseau state that, in many cases where they had suspended a piece of intestine, the blood not only gravitated to its lower parts, but transuded through the mucous coat. In this way blood may be extravasated, and mistaken for the effects of external violence; in the former case, however, it is always fluid, while, in the latter, it is generally coagulated.

Any obstruction to the free return of the blood to the right side of the heart, or to its circulation through the lungs, will cause redness and congestion in parts independent of inflammation. How often do we observe blood effused under the inner membrane of the stomach from this cause, and which has been set down as evidence of other disease? The mucous lining of the intestines frequently exhibits red patches throughout different parts of its course; and even blood will collect within the canal, where no inflammation was present, but where some obstacle to the free circulation through the lungs had existed. There are, therefore, four circumstances to be taken into account in judging of redness in the dead body. These are—the period at which the examination is made, and the probability of transudation—the mode of death, whether sudden or gradual—the position in which the body has been laid—and the presence of any mechanical obstruction to the circulation. Whenever we observe that redness is not confined to one spot, but is exhibited in different places remote from each other, we may rest assured that it arises from some of the causes now mentioned; on the other hand, where it appears solitary, and no other marks of congestion or transudation are present, there is a probability of its being the result of inflammation, and we must be cautious as to the opinion we mean to deliver.

The most unequivocal proof of redness being a sign of inflammation is, where it is found united with some of the common products of the disease. Effusion of serum or blood would not entitle us to come to any immediate conclusion, because they often arise from other causes; but when, along with the redness, we find lymph, or flakes of lymph mixed with a serous fluid, we may then be satisfied that inflammation had been present. Coagulating lymph is thus a certain test of inflammation; and we know of no instance where it can otherwise be formed. It may happen, nevertheless, that time has not been allowed for lymph to be thrown out, and still the other evidence may be so clear, that it can hardly be set aside. If, for example, we are confident, from the symptoms during life, that a certain organ was inflamed; if, on examination after death, redness, more or less intense, remains, and which cannot be washed out like a piece of blood-stained membrane; and if due attention has been paid to position, the chance of mechanical obstruction, and the probability of transudation—we may safely enough infer that inflammation had been present. In fine, whenever we are attempting to ascertain that inflammatory action has been the cause of death, we ought carefully to contrast the previous symptoms with the appearances seen on dissection, and

these latter again with the changes going forward in the body before and after vitality has ceased. Unless this is done, it will be impossible to arrive at the truth; and, in legal medicine, errors, involving the life of a human being, are, from want of necessary precautions, not unfrequently committed.

SECTION IV.

CONSTITUTIONAL EFFECTS OF LOCAL INFLAMMATION.

We come, in the next place, to treat of the general or constitutional effects, commonly denominated the symptomatic fever. While inflammation often proves fatal, by altering or suspending the functions of parts essential to life, much of the danger to be apprehended arises from the febrile disturbance to which we are about to direct our attention. The importance of this subject, in a practical point of view, is thus apparent; while, to the theoretical pathologist, it has afforded much scope for interesting investigation. It is not our duty to enter at length into all the curious details involved in its discussion; but we shall endeavour to give such a sketch as to render the matter intelligible, as far as is subservient to our present purpose.

Of the history of fever, a very brief outline will suffice. Two theories have been brought forward to explain the nature of febrile action—the one ascribing the whole to a morbid change in the composition of the fluids—the other, to some modification in the state of the solids; and from these have sprung the doctrines of the humoral and the nervous pathology. The ancients, nearly ignorant of the structure of the body, and still less acquainted with its functions, ascribed the origin of fever to some change in the condition of the fluids. The greater bulk of these, compared to the solids, and the manifest derangement of all the secretions observed during a paroxysm of fever, tended to confirm them in this opinion. The blood, from its high importance, was the fluid to which their attention was chiefly directed; but its composition, like many others, was then unknown. If a redundant portion of bile made its escape into the vessels, or if the blood became charged with phlegm or other viscid matter, fever ensued, and remained till some alteration was again effected. Hippocrates advanced the opinion, supported afterwards by Galen and his followers, that the system was impregnated with some noxious substance, exciting certain movements and commotions which constituted a paroxysm of fever. He supposed that nature exerted all her powers to accomplish its expulsion; and, according to the length of time required, so was the disease of shorter or longer duration. The struggle which nature made to relieve herself, caused the ebullition or con-

coction; and the more obstinate the resistance from the foreign power, the more violent was the fever which ensued. The changes more especially taking place during the progress of the exanthemata appear to strengthen the conjecture of Hippocrates, that some morbid agent has been received into the system, and it is afterwards eliminated. The doctrine was well received, and very generally adopted for many ages; and however unsatisfactorily it may account for all the phenomena of fever, there is much truth in the assertion that a morbific poison has been applied. The chemical doctrines of Paracelsus and Van Helmont, which set forth that the fluids possessed at one time an alkaline, and at another, acid properties, and that an effervescence took place, producing fever, rendered the humoral pathology still more imposing. Boerhaave, dissatisfied with the opinion of Hippocrates, that there was a change in the composition of the blood, applied his mechanical theory of an *error loci* to explain the cause of fever. Towards the close of his life, and with a candour not often seen among those belonging to our profession, this illustrious man acknowledged the insufficiency of his views, and partly adopted the doctrine of the nervous pathology, which had by this time been brought forward.

So far the fluids had been regarded of paramount importance, and to their modifications were all diseases to be traced. But few theories in medical science, founded on conjecture rather than on close observation and experiment, have been destined to survive; and the humoral pathology, in all its pride, gradually bent before the more refined hypothesis of the solidists. In contemplating the fluctuation in opinion and estimation to which the doctrine of the fluids has been subjected—for several hundred years the idol of its party, then neglected and despised (for such was the rancour of controversy), and now again rising into repute—we have a memorable instance of the imperfection and instability of all human reasoning. In a published lecture on typhus, Dr. Clanny has endeavoured to show that a cessation of sanguification is the ostensible proximate cause of the fever; but such a theory is at variance both with the manner in which the disease often makes its invasion, and likewise with its decline. He considers, however, that the solids are first affected, and that the important changes taking place in the blood are established through them. Dr. Stevens, on the other hand, believes the derangement in the circulating mass to form the first link in the chain of morbid phenomena. In opening the heart, in cases of yellow fever, he found a fluid as thin as water and as black as ink; and, in all parts of the body, the distinction between arterial and venous blood was lost. According to him, the blood undergoes a regular series of changes, first becoming thinner and losing its saline principles, then turning black, and having its vitality so destroyed as to be incapable of supporting the action of the heart. He farther considers that the aërial poisons, from which all pestilential diseases proceed, are carried with the atmospheric air into the lungs, mingle there with the blood, and being circulated

over the body, cause subsequent morbid action of the solids. The fact of the composition of the blood being altered in fever, and many other diseases, will be acknowledged by every practitioner of the present day, whatever value he may feel inclined to attach to it; the difficulty is in accounting for the order of occurrence, whether such changes precede or are acquired during the progress of the affection; as, in the one case, they would be the cause, in the other, only the consequence of an aberration from the state of health.

Andral is of opinion that the morbid alteration in the circulating mass takes the precedence. We find him stating—"The fever, termed inflammatory, seems often to arise from no other source than the blood being too rich in fibrin; in like manner, an impoverished state of the blood, whether accidental or natural, is often connected with mucous fevers, and with those characterised by a sudden sinking of the vital powers; and that the source and primary seat of typhus fevers, properly so called, is the blood, inasmuch as they are caused by the introduction of deleterious substances, such as animal or vegetable effluvia into that fluid." Whatever be the order of occurrence observed, it is certain that, in fever which partakes of an inflammatory character, the blood contains at first a larger proportion of fibrin, and forms a firmer coagulum; while, during the progress of the case, or in one accompanied from the beginning by evident marks of prostration, there is a diminution in the quantity of fibrin, and the clot is soft and loose. We are inclined to adopt the views of Andral with respect, at least, to the inflammatory variety of fever; for we have many opportunities of observing this condition of the blood, in persons in the habit of having venesection performed once or twice a year, and who, unless some evacuation or other is practised, become often the subjects of local inflammation. If we regard the morbid alterations in the composition of the blood as the primary source of fevers, we can easily explain the subsequent derangement in the functions of organs, and the vitiation of the different secretions during their continuance. A want of facts prevents us from arriving at any accurate conclusion; the subject is yet in its infancy; and while the humoral pathology will, we trust, emerge from the obscurity and redicule thrown over it by its bigoted opponents, we, at the same time, hope that its due share in the constitution of disease may only be assigned to it.

In opposition to the doctrine of the humoral pathologists came that of the solidists, first introduced and promulgated by Stahl. He supposed that there existed a salutary agent, the *vis medicatrix*, which, on perceiving any injury done to the body, immediately exerted itself to counteract it. Reckoning this point established, he concluded that fever consisted of a *tonicus spasmus* of the different nerves, to overcome which there was a struggle of the *vires medicatrices*, and which excited those commotions in the system constituting the disease. So imposing was this new theory, that even Boerhaave become reconciled to it, although he considered the change as merely mechanical, and not consisting of any different

state of the living solid. By others, however, it was condemned, and said to be both unintelligible and without proof. Hoffmann afterwards adopted and refined the views entertained by Stahl. The patient investigations of Hoffmann and the results of his inquiries were forgotten, till again revived by Cullen. Like both his predecessors, Cullen believed in the operation of a *vis medicatrix naturæ*; and, as Hoffmann had done, he gave to every part of the body its due share of this salutary power. Thus armed, he gave forth to the world his ingenious theory of fever, one which professes, in some respects, to give a more natural explanation than any other before or since offered, and which, if now falling into neglect, cannot be said to be dying away before a model of more stately structure.

The most unmerited censure has been heaped on Cullen for having made use of the phrase *vis medicatrix naturæ*; but he was not the inventor of this tutelar deity, as many have ignorantly supposed; it had descended from Stahl; nor did Cullen receive it without limitation, for he justly considered it as throwing an obscurity over all our reasoning. By the *vis medicatrix* was only meant the reaction of the system, or the efforts made by nature to restore the several functions of the body, when they have been interrupted or disturbed by some noxious power; and, used in this sense, it was also the language of Hippocrates. Although the phrase has been laid aside by modern writers, they acknowledge the same principle when they refer certain effects to the reaction of the system; and whoever, in the ordinary course of his experience, has had occasion to observe the human body in its opposite states of health and disease, must have recognised the attempts made to regain the one, and to provide against the ravages committed by the other. We cannot, it is true, explain the means by which nature endeavours to accomplish these ends, any more than we can explain how life itself is maintained; we witness the results only, the steps by which they are effected remain hidden. Again, where nature is unable to accomplish her work, or when the reaction of the system is so violent as to threaten destruction to life, the interference of art becomes necessary; in the former case, we endeavour to promote her efforts; in the latter, to preserve them within proper bounds. To this power, then, of self-preservation, whether we choose to know it by the title of *vis medicatrix*, or by that of reaction of the system, do we ascribe many of the changes occurring within the animal economy, both from the application of remedies and the attacks of disease; and it was only to express this original law of our constitution that Cullen sanctioned the use of a phrase about which he has been so ungenerously reviled.

It was a leading feature in the theory of Cullen—and he borrowed the idea from Boerhaave—that one stage of fever was the cause of another, and that the different stages should therefore follow in regular order. As the cold stage occurs first, and gives rise to the warm, it was conceived that, provided the cause of the former

could be discovered, the nature of a paroxysm would be easily explained. From the weakness of pulse, the general coldness of the limbs, the languor, uneasiness, deranged sensations, and impaired animal functions, Cullen inferred that the causes of fever operated by diminishing the energy of the brain, and thereby enfeebling the action of the heart. He attempted to prove that contagion, miasmata, cold or fear, induced debility; that any cause which brought on this would renew a fever after convalescence; and, from such premises, he concluded that fever essentially consists of three states—one of debility, another of cold, and a third of heat. These constituted the general phenomena of a paroxysm; and as, according to his supposition, a regular succession was observed, the debility was the *primum mobile*, creating the cold stage, and this, in its turn, the warm. The Herculean task still remained unfinished. How does the cold stage arise out of debility? Here the theory is imperfect; and we are merely referred to that general law of the animal economy, by which all noxious powers are obviated or expelled. Observing that derangement and suppression of the secretions continued till the sweating stage set in, Cullen imagined that a spasm of the extreme vessels had taken place. He supposed that the spasm formed during the cold stage proved a stimulus to the heart and arteries, whereby their action was increased; and, in order that debility might not be presumed incapable of producing these effects, he attributed the cause of the spasm to the *vis medicatrix*. The extreme vessels were every where in a state of debility, and this formed an important part of the proximate cause of fever. The debility of the capillaries extended from those on the surface to those of the stomach, and caused the anorexia and vomiting frequently witnessed during the cold stage; it arose out of a diminished energy of the brain, from whence ensued the delirium and imperfect sensations formerly alluded to. This state, however, was transient; increased action of the heart and arteries followed, and by which the energy of the brain was restored; the spasm of the extreme vessels was removed, and diaphoresis ended the paroxysm.

The complex theory of Cullen would require a volume to unravel it; and, even were the task accomplished, we should find that we had not advanced a single step towards explaining the nature of fever. It was an essential position that the different stages should succeed each other in regular order; and, if we can show that this is not always the case, the chain of reasoning is at once broken and destroyed. Now it often happens that in intermittents which have become irregular, the order of occurrence is reversed; and, in other instances, as in the variety described by Cains, diaphoresis has accompanied the fever throughout. The idea that spasm of the extreme vessels occurs during the cold stage of fever, and proves an irritation to the heart and arteries, is supported by no evidence whatever; and, again, the supposition that the increased circulation restores the energy of the brain, and thus

removes the constriction of the capillaries, is alike without foundation. There is probably much truth, however, in the statement that the causes of fever operate first and most strongly upon the nervous system; but we never find that the derangement of function so created is repaired by the hot stage, as Cullen wished to impress. Notwithstanding its ingenuity, when applied to explain the nature of fever, the theory is deficient; it has done good, by serving to connect in a more regular manner the different symptoms; and it has done evil, by leading the mind astray to the use of means calculated to remove the supposed spasm of the vessels.

From the celebrity of the author, these views of the nature of fever were very generally received until they were exposed by Brown, at first the friend and afterwards the inveterate enemy of Cullen. The Brunonian doctrine attracted much attention from its professed simplicity, and a long time elapsed ere the principles on which it was founded were acknowledged to be false. Man was now represented to be a mere passive machine, endowed with a certain portion of life, and capable of being acted upon by external agents. The natural period of life was thus fixed; every animal received a certain share, which, if not too rapidly exhausted, would continue for a certain length of time. The phenomena by which life was to be exhibited resided in an uniform indivisible property termed excitability, of itself incapable of producing life, and requiring for this purpose to be acted upon by stimuli. The quantity of excitability allotted was more or less quickly exhausted by the application of stimuli. Should the stimuli be in excess, the excitability was impaired; if deficient it accumulated; a certain portion, however, was necessary for life, for, if totally withdrawn, our existence ceased. Health was attained only when the stimuli and excitability bore a certain relation to each other; but this balance was often subverted, and then disease commenced. The Brunonian classification of diseases was into sthenic and asthenic; the former being of one kind only, the latter comprising cases wherein the excitability was either exhausted or morbidly increased. The treatment was obvious—to employ means either to lessen or to rouse the excitability. The diseases of increased excitement, however, were few, compared with those where debility was manifest, and to the latter, therefore, the Brunonians devoted their principal attention.

According to these views, the due performance of every healthy function was constantly exhausting the excitability originally bestowed upon us; nay, the very processes of digestion and assimilation, which are supposed by sober-thinking people to preserve life, must have been daily cutting short our career. The supposition, that man is a mere machine, endowed with a certain measure of excitability which gradually diminishes and cannot be resupplied, is at variance with all the natural phenomena of sleep, nutrition, fatigue, and muscular exertion; and, consequently, the superstructure raised on such a foundation must be weak and indefensible.

The theory of Brown lingered longer in other countries than in his own. It was carried into Italy by Rasori, one of its most strenuous supporters. A few years afterwards, the petechial fever breaking out at Genoa, gave the disciple an opportunity of following out his favourite treatment, but many of his patients perished. Rasori next tried a controstimulant plan, by which the loss of human life was much diminished; convinced, then, of his error, he discarded for ever the Brunonian principles. This proved a decisive blow to what may, without any hesitation, be named the most pernicious theory ever invented for selfish motives.

Another class of the solidists has assigned to fever a local seat in the body. Marcus and Clutterbuck consider inflammation of the brain to be the cause of fever, and accordingly direct their attention principally to the state of this organ. Pain in the head is the symptom during life which has led to this opinion; but we are not warranted, where the nervous system is so much deranged, in considering pain as always proceeding from inflammation, and we think experience freely justifies us in making this assertion. Other pathologists, again, consider lesions of the intestinal canal as the essential cause of fever. Amongst the most conspicuous of this class stands Broussais, who thinks that the influence of such a doctrine over the population would be even more favourable than vaccination. The same views have been supported by Petit, Serres, Bretonneau, and Louis, the latter of whom has given an elaborate account of the different lesions occurring in fever, and confirms the dissections previously made by Bretonneau, that the primary source of disease is in the mucous glands of the lower portion of the ilium and the cœlum. Agreeably to the French pathologists, every species of fever proceeds from a *gastro-enterite*, and which may give rise to other symptoms during its progress from bad to worse. Such lesions would seem to be more frequently met with in some countries than in others, arising apparently from local causes, as indeed the effects of fever vary at all times according to climate, season, and particular type which may be prevailing. While we willingly admit the frequent complication of fever with inflammatory action, and the urgent necessity that exists for watching the invasion of the latter, we would be understood as plainly to deny that fever was dependent on local disease. It is, no doubt, true that inflammation of particular organs will co-exist with or supervene on an attack of fever; but it is equally true that fever may arise and go through its whole course without any such local derangement. We shall afterwards assign reasons for our belief. In the meantime, we may observe that both theories have done good, by putting us on our guard against these local attacks, and leading to a more successful line of treatment; but no better argument against the exclusiveness of such views could be given, than the difference existing among their supporters as to the proper seat of disease.

Again, fever has been regarded as an essential disease not dependent on any local affection, but frequently complicated with such,

and which constitutes the greater source of danger. To this class of reasoners the name of *essentialists* has accordingly been given. Fordyce, in our own country, considers fever "as a general disease which affects the whole system, the head, the trunk of the body, and the extremities; the circulating, absorbing and nervous systems; the skin, the muscular fibres, and the membranes; the body and likewise the mind." Pinel of France so far takes the same view, but he has evidently become a *localist*, when he assigns a seat for the different varieties, as the inflammatory in the organs of circulation—the bilious in the mucous membrane of the small intestines, &c. For our own part, we think that all attempts to refer fever to an affection of one individual organ or system are both fruitless and erroneous; and that, when sober reasoning comes to be substituted for the erratic excursions of the imagination, the fluids as well as the solids will be allowed to participate in the general derangement. Whether the primary source of common fever consists in a morbific poison applied to the blood in the lungs, and by which the nervous and other systems are subsequently affected, or whether the changes in the latter take precedence of the changes in the circulating mass, we do not pretend to determine; but we hold that the records of established cases coincide with daily experience in proving the worst varieties to occur without the coexistence or supervention of local disease. It is by reference to the great book of nature, the same through a thousand editions, that this practical point must be determined; and it is to the interpretation of her work that the life of the physician should be steadily devoted.

In attempting to define disease, the object to be kept in view is to give an enumeration of those symptoms which are deemed essential to its existence, and without the manifestation of which it could not be said to have occurred. In regard to objects of natural history, where each individual has its progenitor exactly the same as itself, the business of defining is precise and limited. In the science of medicine, far greater latitude must be allowed, for there we cannot classify diseases according to their real nature, but only by the symptoms to which they give rise; and when the greater number of these are observed, the particular morbid state is then inferred to be present. Boerhaave attempted to reduce the phenomena of fever to shivering, increased frequency of pulse and heat; but this attempt at generalisation fails, for many diseases which we consider febrile, not only want these, but are attended by opposite symptoms, and several others would require therefore to enter into the definition. The concourse and succession of symptoms are briefly given by Cullen in these words—"*post horrorem, pulsus frequens, calor major, plures functiones lesæ, viribus præsertim artuum imminutis.*" These he illustrates at greater length in that part of his Practice of Physic which treats of fevers; and, although sometimes defective, his descriptions of disease indicate a close observance of nature, and are well worthy of the attention of the medical student.

Some degree of languor and a sense of debility usually usher in febrile affections—premonitory signs, be it remarked, which are far more generally seen in what is termed idiopathic than in symptomatic fever. These feelings are succeeded or accompanied by a coldness over the body, and shivering to a greater or less extent. The sense of cold then gradually subsides, and a preternatural heat is next experienced by the patient. During these changes the *plures functiones lesæ* manifest themselves in some form or other. The pulse, at first small and quick, rises in fulness and strength, the natural secretions are suspended or altered, and the functions of the nervous system impaired. After a while the increased circulation abates, the preternatural heat diminishes, and a solution of the fever takes place in the way of some critical evacuation, commonly as perspiration, diarrhœa, or hemorrhage.

Such may be considered as a brief statement of what takes place to a greater or less extent in febrile affections; and it is to be regretted that we have no more concise way of expressing our knowledge of their existence. We have seen enough to satisfy us that the word fever does not convey a simple idea, but is employed rather to denote a certain assemblage or succession of symptoms which cannot be comprehended under any single term. It is a remarkable circumstance, also, in the history of these diseases, that, although we cannot state precisely by what symptoms they are known to exist, to the eye of the experienced practitioner, a fever patient presents an object which is immediately recognised. In short, we are perfectly familiar with the appearances of fever, and can give to it without farther examination its proper name; yet it is with considerable difficulty we can assign a reason for our belief.

The whole course of the symptoms, from the commencement of the rigours to the close of the critical evacuation, is termed a paroxysm. Sometimes the fever consists of a single paroxysm; sometimes of a number of distinct paroxysms, with complete intermissions between them; sometimes of a protracted paroxysm with only very slight remissions and corresponding exacerbations. On the occurrence of these is founded the division of fevers into intermittent, remittent, and continued. It will likewise facilitate our description, to remark that the phenomena of continued fever present varieties which give rise to a subdivision of it into sthenic or inflammatory, and asthenic or typhoid. The nature of the affections so designated need not be explained till we proceed further in our investigation; all that we wish at present to be understood is, that these varieties depend upon certain peculiarities in the predominant symptoms—that the general character of the inflammatory or sthenic is an exaltation of the powers of life—while the typhoid or asthenic is recognised by marks of debility, depression, or diminution of strength. We shall form, also, a better idea of the subject under discussion, by considering the symptoms as they relate to the different functions or systems which are deranged. We do not enter into the question, in what order are the various

functions affected? and do the successive functional derangements stand to each other in the relation of cause and effect? We are merely desirous of taking a view of the phenomena occurring in fever.

Symptoms indicating derangement of the nervous system are, then, among the earliest that appear in febrile diseases, but our present knowledge does not enable us to say on what this functional derangement depends, nor is it likely that we shall ever arrive at any accurate conclusion on this subject. Of all the changes observed in fever, those happening in the nervous system are the most difficult to comprehend, or to which we can refer, with least certainty, the proper symptoms which spring from them. A degree of confusion of thought, a want of intellectual energy, certain indescribable sensations with a feeling of *horripilatio* (for we have no English term so expressive) over the body, constitute the first symptoms complained of by the patient. The most prominent change referable to the nervous system—and one generally met with in all diseases entitled to the name of febrile—is a feeling of listlessness or an uneasy sensation quite peculiar to this state. No description, to use the language of Dr. Southwood Smith, can convey any idea of it to one who has not felt it, and to him who has, the word fever recalls that feeling so instantaneously and so vividly, that most unprofessional persons conceive that it is this very feeling which constitutes the essence of the disease. It is much more distressing than pain; the mere restlessness which accompanies and forms so large a part of it, any one would gladly exchange for intense pain. In all diseases it is this which makes the sufferer on his midnight pillow exclaim, "O! that it were morning!" and, in the day, "would that it were night!" To this feeling, also, we may add the pain experienced in the back and limbs. The headache so generally present in fever is commonly a symptom merely of the general nervous derangement, although at other times dependent on dangerous inflammatory action within the cranium. The sensorial powers are always affected; and we have every variety present, from slight confusion of thought to delirium or complete aberration of the mental faculties.

In the inflammatory or sthenic form of fever, the nervous symptoms are less prevalent; while in the typhoid or asthenic, they constitute the most prominent feature. So much is this the case, that the latter variety is not unfrequently denominated nervous fever. The delirium observed in fever usually appears under two forms —the delirium ferox or vehement species—and the delirium mite, typhomania, or low muttering; the first accompanying the sthenic, the second, the asthenic type. It is from this symptom of asthenic fever that the word typhus is originally derived, τυφος, literally meaning stupor. The functions of the nervous system, in reference to the muscular powers of the body, are likewise remarkably affected. Cullen has noticed this fact, in his definition of the prostration of strength, by the expression "*viribus artuum præsertim*

imminutis;" and, as a symptom of fever, it holds a very prominent place in the more severe forms of typhus. In connection with this symptom, also, we have the tremour of the tongue and limbs, and the *subsultus tendinum*. In the inflammatory fever, the premonitory signs depending on derangement of the nervous system are much less distinctly marked, indeed often entirely wanting; there is a preternatural exaltation of the powers of the system, and the muscular energy appears for a time to be increased.

The next system affected, and that probably, too, in the order of occurrence, is the circulating. A preternatural frequency of pulse is one of the most prominent symptoms of fever. We have already hinted that this is not always nor necessarily present, as there are cases which, from the other signs, must be regarded as febrile, where the pulse is either not accelerated or is slower than natural. In that form of fever, the description of which we have more particularly in view, the pulse is almost always preternaturally quick. Quickness of pulse, it will be recollected, is, to a certain extent, only a relative term, and bears reference to the usual number of pulsations in the individual when in health. For example, in a young person the pulse may be habitually from eighty to eighty-five in a minute; but, in an old person, whose natural standard does not exceed sixty or sixty-five, this would be regarded as an increased acceleration. Reference must also be made to the idiosyncracy of the individual. In speaking of the pulse, we may observe that not only the number but likewise the character of the pulsations must be carefully noted. In the commencement of fever, or in what is called the cold stage, the pulse is weak, quick, and generally irregular. As this stage subsides, and the warm approaches, the pulse acquires a fuller, harder, and more bounding character, and may continue so till a solution of the fever takes place. The pulse, however, is not always full and strong, it sometimes continues weak or becomes so, and this is one of the distinctive marks between fever of an inflammatory or of a typhoid type. The sthenic variety is usually characterised by a full, hard, or bounding pulse; the asthenic, on the other hand, exhibits the small, weak, although sometimes sharp pulse. It is not our business to discuss the varieties of the pulse or their causes, we shall merely observe that we measure its strength or weakness by the quantity of blood circulating through the vessel, and the greater or less pressure required to suspend the pulsation. Both these conditions, again, may be coupled with, or occur without, that peculiar state—difficult to express in words, but easily remembered when once felt—to which the terms sharpness, hardness, or wiriness have been applied.

Connected with the derangement occurring in the circulating system, we may notice the difference of temperature observed in fever. As there is reason to believe[1] that the generation of heat is chiefly dependent on the function of the circulation and the

[1] See Local Symptoms—Animal Heat.

changes going forward in the mass of blood, it is natural enough to expect that an alteration in the animal temperature will be effected. A good deal of variety exists in this symptom; but, even even at the highest, the absolute temperature is not so great as we might be led, from the feelings of the patient, to expect. In order to measure the degree of heat, the thermometer should be applied to the arm-pit, or under the tongue, and, when the observation is made in this way, it will be seldom found to exceed 107° F. In some species of fevers, as the exanthemata, the heat will be as high as 112°; but, in common fever, the rise of temperature seldom goes beyond 7°, assuming 100° as the natural standard.

Amongst the derangements, also, exhibited by the circulating system, we ought to mention the regular distributions of blood which so frequently take place, and which have been described by pathologists under the name of determinations. These, in common fever, are observed to occur in some of the great cavities of the body, and demand particular attention, both from the symptoms immediately arising from them, as well as the tendency to inflammatory action thereby induced. Probably nothing is so common, during the progress of fever, as irregular supplies of blood to different organs, and nothing we know of predisposes more to local complications; but such determinations are met with principally in idiopathic fever, and not in that variety connected with inflammation.

The most important change, in the system we are now speaking of, remains to be noticed—the alteration in the character and properties in the circulating mass, so conspicuously seen in sthenic or inflammatory fever. In point of importance, we may look upon the derangement in the blood and its circulation as paramount; and by many we should not be thought to err, were we to attribute to them all the other changes which arise in fever. When blood, drawn during inflammation, has coagulated, its surface, instead of presenting the usual florid or scarlet hue, is found covered with a white, opaque, adhesive matter, strongly resembling coagulated albumen. To this appearance has been given the name of the buffy coat, the *crusta coriacea* of some authors. The immediate cause of this change is the precipitation of the red globules to the bottom of the vessel, and by which the fibrin or lymph above is deprived of its colouring matter. When the surface of the coagulum is rendered hollow or concave, the blood is then said to be cupped; and in addition to this, we often find its edges puckered or fringed.

The buffy coat of the blood varies in thickness from a line to one or two inches, supposing the fluid to have been received into a common tea-cup. The coagulum, when divided from above downwards, resembles nearly the section of an egg, with the small end of the oval placed uppermost. It is much more dense and firm than the coagulum of ordinary blood, the density and cupped appearance being usually in proportion to the intensity of the

inflammatory action, and the strength of the constitution. These phenomena are more conspicuous in some cases than others, and particularly where lymph has not been effused as a product of inflammation. In consequence of the unusual firmness with which the particles of the buffy coat cohere, the serum is more effectually squeezed out; hence the coagulum appears proportionally smaller from the quantity of serous fluid with which it is surrounded. There is, however, no increase in the quantity of serum; there is only a more complete contraction of the clot. When blood is slightly buffed, or, as it has been termed, sizy, the serum appears less in quantity from being detained within the mass, while in reality it is increased.

When the coagulum of inflamed blood is examined, it is found to consist of pure fibrin, containing a small quantity of serum. The late Mr. Thackrah[1] considers the buffy coat as a network of fibrin, enclosing a portion of serum, and has shown experimentally that it is made up of 18 per cent. of dry solid fibrin, 26 per cent. of dry solid matter from the serum, and 56 per cent. of water. The dry solid matter to which he refers is albumen, always found in much greater quantity in the serum of inflamed than in that of healthy blood. No two analyses of the buffy coat will be found to agree; for the proportions of the blood in diseases vary in different individuals, and in the same individual at different times, in the same way as they do in a state of health. It has been supposed that only venous blood, when drawn, puts on the buffy appearance, and this has been assigned as a reason why, in certain cases, the *crusta coriacea* is wanting; but Dr. Tweedie has seen blood taken from the temporal artery buffed and cupped in two separate instances; one where the patient laboured under inflammation of the chest, the other where the cerebrum was affected during fever; and the same thing has been observed by Gendrin, and by the late Dr. Gordon, of Edinburgh.

The modification, then, which the blood seems to undergo in inflammatory fever, consists in an increase of its fibrin and the animal matter contained in the serum. Gendrin has very minutely detailed the appearances presented by the blood under the different varieties of inflammation, and the relative proportions which the crassamentum and the serum bear to each other. In very acute inflammation, the buffy coat is firm and elastic, of great thickness, and much cupped; the whole of the serum is nearly squeezed out, and in point of volume, the clot appears to this fluid as one to one and a half and sometimes two. In some cases, the proportion of serum is less than the crassamentum, the latter adhering to the bottom of the vessel in the form of a truncated cone. From the firm contraction of the coagulum, however, the serum generally seems more abundant than it really is, and, in such cases, appears usually colourless and free of red matter, even in the lower part of

[1] See his excellent Essay on the Blood.

the cup. In less vehement inflammation, the buffy coat is cylindrical in shape, does not extend so deeply, and is not so strongly cupped; while the serum equals twice the volume of coagulum. In subacute inflammation, the blood presents the sizy appearance merely; the buffy coat extends only one or two lines in depth, and is of less density and slightly ovoid; the serum, which at least equals twice the volume of coagulum, is viscid and limpid above, but reddish at the bottom of the vessel, from the precipitation of the coloured globules. These phenomena are varied according to the seat and intensity of the inflammatory action, and many other modifications might, no doubt, be described.

The change effected in the circulating fluid has been a subject of keen discussion among pathologists. The immediate cause of it is a more complete separation of the serum, and a precipitation of the red globules, leaving the colourless fibrin contracted into a firm dense mass; the remote cause is involved in much obscurity. The coloured particles being found to be specifically heavier than the other ingredients of the blood; and it having been observed that the time, in general, required by buffed blood to coagulate exceeds that of healthy, it naturally occurred that the appearance in question depended on the mechanical separation of the red globules merely from their greater density, just as sand and water separate after being agitated together. The circumstance of slower coagulation, we may simply remark, is not found to be an uniform antecedent to the formation of the buffy coat; neither does it appear, even were it so, to afford a sufficient explanation of the other phenomena accompanying the process. Gendrin maintains that coagulation commences sooner, and is more speedily completed in inflamed than in healthy blood, and with a rapidity proportional to the quantity of the buffy coat. The same thing has been amply proved by Dr. Stokes,[1] and may be witnessed by any one in the ordinary routine of practice. It has been also imagined that the colouring matter, as well as the serum, are squeezed out, by the fibrin contracting and cohering with greater force than in health; but this is stating the effect without assigning the cause.

It is scarcely possible to give any explanation of the formation of the buffy coat till the cause of coagulation in healthy blood has been first agreed upon. The fluidity of the blood while circulating in the vessels, and its coagulation or spontaneous separation when withdrawn from the body, are circumstances which cannot be explained by any chemical or mechanical agency; and, perhaps, we commit the least error by regarding them as ultimate facts in the animal economy. Neither heat or cold, rest or agitation, although all exerting an influence over the time in which coagulation takes place, have any other share in the process whatever. Nor, as Sir Charles Scudamore[2] has attempted to show, does coagulation depend on the escape of carbonic acid. If we surround

[1] Pathological Observations.

[2] Essay on the Blood.

a portion of blood with an atmosphere of carbonic acid, its upper surface is merely prevented from assuming a florid colour; we do not, in the slightest degree, retard coagulation. If we place blood in the receiver of an air-pump, and exhaust the air, by which the escape of carbonic acid would be abundantly favoured, we do not cause more speedy coagulation. We have also sufficient chemical authority for denying that carbonic acid makes its escape in such quantity as to account either for the celerity or tardiness with which the separation takes place.

Mr. Thackrah has concluded, from his own experiments and those of Sir Astley Cooper, that the coagulation of blood, under ordinary circumstances, depends principally on its separation from the living vessels; and that its remaining fluid, in cases of sudden death, where the nervous energy over the body is destroyed, arises from its vitality not being extinguished. In such instances, the blood remains fluid for several hours, and then slowly coagulates after being removed. There is a natural tendency in the fibrin to coagulate, and which is prevented, no doubt, by its motion in the vessels; but that a certain vital relation between these is established, is apparent from the fact that blood coagulates speedily in a dead vessel, while it remains fluid for some hours when enclosed between two ligatures in the living body. It was these, and such like phenomena, which led Hunter to speak of the life of the blood, understanding the word life to imply a series of changes, unlike those which take place in unorganised matter. In the same manner, we may state, that there is a vital relation subsisting between the fibrin and the colouring matter, and which undergoes alteration in disease. We cannot attribute the formation of the buffy coat to any of the circumstances attending ordinary coagulation; neither can we ascribe it to the fibrin being thinner, as has been presumed; for we find that, when the separation has taken place, the mass is unusually firm, and coheres with greater strength. There seems, indeed, to be a repulsion exerted between the colouring portion and the fibrin, or, at least, there is an unusal tendency to separation in those cases where the buff appears. Two circumstances observed during the process of coagulation, lead to this conclusion, and we may give them in the words of Dr. Alison.[1] "First, the formation of the buffy coat (though no doubt favoured or rendered more complete by slow coagulation,) is often observed in cases where the coagulation is more rapid than usual; and the colouring matter is even observed to retire from the surface of the fluid, in such cases, before any coagulation has commenced. Secondly, the separation of the fibrin from the colouring matter, in such cases, takes place in films of blood so thin as not to admit of a stratum of the one being laid above the other; they separate from each other laterally, and the films acquire a speckled or mottled appearance, equally characteristic of the state of the blood as the buffy coat itself."

[1] Outlines of Physiology.

Admitting, which we freely do, our inability to explain the manner in which the buffy coat is formed, it becomes an interesting question how to account for its presence. This is a point still uninvestigated by pathologists. Mr. Thackrah represents the fibrin, which in a state of health goes to repair the muscular and other fibrinous tissues, to be increased from more extensive formations, and especially from the occurrence of adhesive inflammation. To this we reply, that, in cases where lymph is thrown freely out on the surface of membranes, as in inflammatory affections of the pleura or peritoneum, the buffy coat is less firm and copious than in those where no albuminous matter is secreted, as in rheumatism. It might hence be inferred that the presence of the buffy coat in the blood was to be ascribed to the suspension of the process of nutrition, rather than to any increased formation of fibrin. But, although the density and abundance of the buffy coat may be augmented, in cases where no fibrin is thrown out as an effect of acute inflammation, as in rheumatism, and lessened in others where this takes place, as on the surface of serous tissues, there are several facts which lead us to the belief, that this change in the blood precedes any suspension of the process of nutrition, and is effected by some vital action of the coats of the vessels. In individuals of sanguineous plethora, as proved by the observations of Ratier and Belhomme, and in persons in the habit of being bled, we have seen the inflammatory appearances assumed by the blood, although no local action had been established; and it is this change in the composition of the mass which, in these instances, so frequently leads to disease. It is well known, also, that, during the period of pregnancy, the buffy coat is exhibited in the blood, and here the process of nutrition is certainly not suspended. Again, the sudden transitions in the mass of blood, show that the vessels have some power in modifying the composition of that fluid. We thus observe that, during blood-letting, where the stream is equable and every other circumstance favourable, the first cup contains more fibrin than the second, and the second than the third; and this change is usually commensurate with the amendment of the patient. Rapidity of coagulation, as Mr. Thackrah has observed, is in the inverse ratio of the quantity of blood left in the vessels; for of quantities, drawn successively under the same circumstances and from the same individual, those taken last coagulate in the shortest time. The short period in which coagulation takes place being generally considered unfavourable to the development of the buffy coat, might be presumed sufficient to explain the difference between the first and last cups of blood drawn in inflammation. We may remark, however, that rapidity of coagulation is chiefly exhibited in animals losing large quantities of blood, or in those perishing from hemorrhage, and does not apply to cases of ordinary blood-letting in the human subject. We have, moreover, direct testimony[1] that buffed blood

[1] See Gendrin's Treatise, and Stakes' Pathological Observations.

coagulates in less time than healthy; and to this we may add the fact that the quantity of fibrin in the second or third vessel, as compared to the first, is diminished—showing a real change in the amount of the solid contents.

Mr. Thackrah has further represented the phenomenon of the buffy coat to be commonly, though not always, connected with rapidity of circulation, and has inferred that, wherever there is a sudden acceleration of the circulation, there will be an equally sudden production of size. The blood of the horse thus occasionally exhibits the buffy coat immediately after a hard gallop, as, in such a case, the contractions of the heart are very rapid. But there are many cases of inflammation, and especially of the substance of the brain, where the buffy coat is very dense and constant, and where the pulse falls even below the natural standard; and, as has been ably observed, it would be requisite, first, to establish the proposition that, when the heart contracts one hundred and twenty times in a minute, it expels from its chambers the same quantity of blood which it does when it contracts only sixty or seventy times. There is, indeed, no ground for believing that, at each of the numerous sudden contractions, the heart throws out the same amount of blood as it does in its more tardy and natural mode of acting; but, on the contrary, there is reason to believe that it expels a much smaller measure, and it might be shown that, though the heart contracts with preternatural frequency, the blood in reality moves much more slowly than natural.[1] It is by no means, however, proved that accelerated or slower motion of the blood is connected with the phenomenon of the buffy coat; and it seems probable, as far as we can judge, that it can take place during the presence or absence of either. We are inclined to attribute the formation of the buffy coat to some vital action of the coats of the vessels. We think the sudden transitions in the mass of blood are best explained by the increase or loss of vital power in the vessels and over the system at large; first, because these changes vary with the state of the constitution as to strength or otherwise; secondly, because they are best seen where debility becomes manifest, and not where bleeding is borne well; and, thirdly, because, as Gendrin has found, blood drawn in recovery induced by syncope, has not only lost its inflammatory character, but the clot is also softer.

The changes thus effected in the circulating mass appear to constitute a necessary and essential part of the process of inflammation, which, when complete, may be described as made up of a peculiar state both of the solids and fluids. But although we have stated that, in individuals of plethoric habit, the buffy coat is occasionally exhibited, and seems, when present, often to lead to local complications, there are reasons for believing that the changes induced in the blood are, in general, only secondary, and caused by the previous morbid incited action of the vessels. Of these the

[1] See Review of Thackrah in Edinburgh Medical and Surgical Journal, July, 1835.

following may be adduced.—First, there are many cases where the buffy coat is wanting, and others where it only appears to a slight extent compared to the local symptoms. Secondly, when the process of inflammation in the transparent parts of an animal is watched with the microscope, the earliest changes seen consist in an increased action of the vessels; and it is not till the local disorder has lasted for some time and reached a certain stage, that the alteration in the mass of blood takes place. Thirdly, if inflammation depended on the changes induced previously in the blood, and not on the incited action of the vessels, it should commence and continue so long as the former state remained; whereas the general rule is, that it subsides as soon as relief is given to the local disorder. Fourthly, in many cases inflammatory action declines spontaneously after it has been fairly established, as we witness in experiments on animals, and in the greater number of these the alteration in the mass of blood has not commenced. Fifthly, inflammation is often the result of violence done to a part, and wherein it is clearly shown, by the symptoms which ensue, that the buffy coat follows the incited action of the vessels. Sixthly, inflammation arises in habits the very opposite to the sanguineous, and where there is rather a deficiency than an excess of fibrin in the blood; moreover, the buffy coat is found, as we shall presently see, in cases where no particular local disorder is present; and from which cause it has been too hastily set down as no indication of inflammation. For these reasons we view, with the exception we have mentioned, the formation of the buffy coat as consequent to the change of action in the vessels; and we farther consider it as a strong proof of the vital relation subsisting between the latter and the mass of blood.

There are several contingent circumstances which have an influence over the formation of the buffy coat in blood drawn from the body, and which require, therefore, to be taken into account before judging of it as a sign of inflammatory fever; these, however, are so uniformly adverted to, in all elementary works on physiology, that it is needless to discuss them here at any length. Thus, the buffy coat is best exhibited when the blood is received into a vessel of ordinary depth, as a tea-cup, and then allowed to remain at rest till the coagulation is complete; when blood is collected in a broad flat vessel, or kept in a state of agitation for some minutes after it is drawn, no separation of the colouring matter takes place, even although intense inflammation should be present. The greater the rapidity with which the blood flows from the orifice in the vein, and the less the distance which it has to fall into the receiving vessel, favour the formation of the buffy coat; on the contrary, when the blood trickles slowly from the arm, or is allowed to fall from a height of three or four feet, no buffy appearance will be indicated. The size of the opening made in the vein, and the rapidity with which the blood flows, frequently cause one cup to be more buffed than the others; and, when the last cup has this

appearance and not the first, it will be generally found owing to the blood having trickled away at the commencement. The degree of temperature maintained during coagulation is likewise worthy of notice; thus, extreme cold, as seen by surrounding the blood with a frigorific mixture, prevents the formation of the buffy coat. The addition of certain substances, as caustic potash, prevents also the separation of the colouring matter; but these can never operate in an accidental way.

Peculiar states of the constitution give rise to the same alteration in the mass of blood as is observed in inflammation. Thus, it is found in pregnancy, in sanguineous plethora, and in individuals under the influence of mercury; it has been seen also in scurvy, and in hysteria where the circulation is rapid. Neither is the density or abundance of the buffy coat any indication of the danger to be apprehended; because, as already stated, it is more conspicuous in inflammation of some fibrinous tissues, as in rheumatism, than where the substance of organs is the seat of disease; and because, in other cases, it is either wanting, or out of all proportion to the existing symptoms. These considerations, while they deter us from regarding the buffy coat as a positive indication either of the presence or intensity of inflammation, point out, at the same time, the impropriety of abstaining from repeated blood-letting, where other circumstances combine to render it necessary. On these points more ingenuity than sound reasoning has been displayed; for where due attention is paid to the influence of adventitious causes, we must be permitted humbly to say, that our own experience has led us to look on the buffy coat as a valuable, though certainly not an unequivocal, sign of inflammation.

Now, this condition of the blood is only observed in fever of the inflammatory or sthenic type, and contrasted with it, we have the appearances presented in the opposite form of the disease. In typhoid or asthenic fever, complicated with local inflammation, the blood either remains natural, or, when it has coagulated, resembles a mass of dark red jelly. In some of these cases, as the fever assumes more of the sthenic variety, the blood shows a tendency to form the buffy coat, a fact which, we think, forcibly points out the vital relation subsisting between the vessels and the fluid which moves within them. In extreme instances of typhod fever, without any apparent local complication, blood, when imprudently drawn, shows little tendency to coagulate, and seems almost destitute of fibrin; when coagulation has taken place, the mass is soft and of a greenish or slightly yellowish hue, the serum is turbid, and the whole has a dissolved appearance, with an unusual disposition to putrescency.

We have taken a brief survey of the derangement observed in the two great systems—the nervous and the circulating. The other functions of the body, dependent, as physiology demonstrates to us, on one or both of these, are likewise affected; and it is necessary that we should run over the principal of them shortly. The

organs connected with the function of digestion are prominently disordered in all febrile diseases. The tongue is furred, and the appearance of this organ is generally characteristic of the type of the fever. In the inflammatory variety, the tongue is covered with a yellowish or whitish fur, principally occupying the sides, middle, and back part of the organ; in the typhoid form, the tongue is dry and glazed, often enveloped in a thick dark crust, and the lips and teeth covered with black sordes. As the febrile disturbance declines, the fur or crust on the tongue separates, leaving the organ again in its natural state. The condition of the tongue is often erroneously considered as an index to that of the stomach, and for this no other reason is assigned than the respondence sometimes observed between them. The tongue has no more particular connection with the stomach than with any other part of the alimentary canal, and this merely because it is lined by a continuation of the same mucous membrane. If the state of the tongue was an index at all times to that of the stomach, every disorder of the one should affect the other; but there are many instances where this is wanting. Emetics have no effect in disordering the tongue, neither in many dyspeptic cases, is there any alteration in this organ. The tongue, on the other hand, is frequently furred in diseases in which the stomach is not implicated, or in which its function may be only impaired in common with that of other organs. Furred tongue is generally the sign of febrile disturbance in the system, in which derangement the stomach likewise partakes, for the desire of food is wanting, and the digestion impaired; but the fever is the cause of the disorder in the one organ as well as in the other. This is a point of great practical importance, as leading to a more rational method of treatment, and especially in cases of dyspepsia, where patients are made to swallow tonic and bitter remedies without discrimination.

The functions of the intestinal canal are always deranged in fever, the secretions are vitiated, constipation or diarrhœa is present, or alternates with each other. We abstain from saying any thing farther here, as to the frequent existence of inflammatory disease within the abdomen, or ulceration of the mucous membrane as the cause of fever, because such is confined to the idiopathic variety, and does not occur in that form which it is our business at present to investigate. The different secretions and excretions are all more or less altered. The urine is diminished in quantity and high coloured, and deposits a lateritious sediment as the fever declines. The perspiration is suspended, and the skin is hot and dry. To the same deranged state of the mucous membrane of the tongue and fauces we ascribe the thirst which harasses the patient, and forms so distressing a part of his disease; and we observe that, as the tongue becomes hard and dry, so is the desire for drink generally increased. The secretion from the liver is also vitiated, being sometimes increased, at other times diminished. The respiratory function is usually disordered; the breathing is either quick

or slow and laborious, and frequently, from accidental complication, we have cough. The more frequent inspirations and expirations, when independent of local inflammatory action within the chest, are simply owing to the increased rapidity of the circulation creating a necessity for fresh air. All these derangements in the nervous and circulating systems, and in the functions dependent on them, as the secernent, digestive, and respiratory, are to be found, more or less, in every case of fever. It is not necessary that they should be all present with the same degree of severity, for this varies in individual cases, and according, too, with the type or variety of the fever; but, making every allowance for these shades of difference, more easily remarked than explained, we consider the febrile condition denoted by the disturbance in the healthy functions we have now pointed out. Were we treating of continued fever as a part of the practice of physic, we would detail more minutely its different forms; we would speak of the various morbid appearances discovered after death; we would enter into the reasonings about its various causes; we would describe its different stages or periods, as that of invasion, formation, and decline; and we would discuss the doctrine of crisis or critical days. But all this, however interesting and necessary to be known, is quite foreign to our present purpose, and we accordingly proceed to state the connection between fever and local inflammation.

When considering the local symptoms of inflammation we stated that their severity depended on several causes, as the intensity of the morbid action, the structure affected, and the idiosyncracy of the patient. Now, should these symptoms occur to any extent, or should inflammation be established with any marked degree of severity, we find that, in a short time, the phenomena of fever are observed. If the local disturbance is moderate, the only striking febrile symptoms may be heat of skin, quick pulse, increased thirst, and furred tongue. The degree of fever is usually proportioned to the severity of the local affection, but it is likewise materially modified, as we shall presently see, according to the structure and importance of the part which appears to be the seat of disease. This is a valuable pathological fact, as frequently the only satisfactory proof of the existence of inflammation, which, from being internal, cannot be seen, is the concomitance of well-marked febrile symptoms with disordered function. We accordingly find that Cullen has commenced his definition of the *phlegmasiæ*, or inflammations, with "*febris synocha*," that is, inflammatory fever; but the definition is incorrect, in as far as the variety of fever understood by the term *synocha*, only occurs in certain cases.

We have endeavoured, as we went along, to point out the distinction of febrile diseases into inflammatory and typhoid, in order that we might be able, at this period, to state more freely the following fact, namely, that certain inflammations are accompanied by sthenic, and others by asthenic fever. Of the former kind are, inflammatory affections of the serous and fibrous tissues; of the lat-

ter, those of the cellular, mucous, and cutaneous. This appears to us a very extraordinary circumstance, and one concerning which no just explanation can be given; it is, at present, to be regarded as an ultimate fact in pathology—an original law of our constitution, to which we must attend particularly when investigating morbid action. Of the necessity or importance of watching the constitutional symptoms induced by inflammation, we may state, that these as frequently prove fatal as the local disease itself; and the fatal event is to be attributed to the depressing effect produced on the heart, or, in less ambiguous terms, to the strongly-marked typhoid character of the fever. Acute inflammation of the intestines may thus prove destructive within three or four days, and that of the pericondium and heart, in less than twelve hours from the commencement of the attack, and, in either, before any change of structure or local cause of death has ensued; hence, inflammation may be fatal, merely from the effects it is capable of producing in the system. Many other instances might be given, but it is needless to multiply examples of what a very little attention to the phenomena of disease will soon render familiar.

By what law, then, or in what way does febrile action arise in the constitution from the presence of local inflammation? It being universally admitted that inflammation has a tendency to spread from one part to another, and especially to textures of a nature similar to that in which the action commenced, it has been inferred, that the morbid condition of the blood-vessels is propagated along their tunics, until the whole of the circulating system is ultimately involved. There are, however, strong reasons for believing that the nervous system is very much concerned in producing the phenomena in question; and the following arguments may be stated in confirmation of this opinion:—First, febrile action is peculiar to organised beings with nervous systems; the diseases of vegetables are purely local. Secondly, we find the fever to be more particularly developed and more readily induced in individuals in whom the nervous system is known from other circumstances to be most prominently susceptible; hence, we observe different degrees of constitutional disturbance following the same amount of local inflammation in different habits. Thirdly, the suddenness of the febrile attack is more like the effect of some nervous sympathy than the gradual spreading of disease; moreover, we have all parts simultaneously affected, and not those first that are contiguous to the inflamed organ. Fourthly, the degree of the fever is not proportioned to the degree of the inflammation without reference to the part inflamed; some structures cause more constitutional disturbance than others, although the intensity of the inflammatory action is equal or even less, and these we find to be connected to the rest of the system by the greater number of sympathies. Lastly, in inducing inflammation for the purpose of experiment, we observe that the first action of a stimulus is on the nerves of the local part, whereby pain is produced, and that from this increased sensibility

all the other phenomena seem to ensue. It appears, indeed, that the fact of febrile symptoms being established, identifies itself with many others, both in physiology and pathology, which are said to be sympathetic; and this is the most satisfactory explanation we can offer of their mode of origin.

In considering the nature of the febrile action set up in the constitution by inflammation, the question has been agitated, Whether the fever is the cause, the concomitant, or the consequence of the local disease? The occurrence of febrile symptoms in some cases, as in measles, scarlatina, or the other exanthemata, previous to any local affection, is no adequate reason for assuming that the fever is the cause of the inflammation; for these eruptive complaints are different in their origin, nature, and progress, from ordinary inflammatory diseases. Again, the frequent observation of marks of inflammation, in persons dying of fever, has led some pathologists to conclude that this latter affection never occurs without an antecedent, whether we can trace it or not; and, to a doctrine of this kind, as formerly stated, we find attached names of the highest respectability, such as Clutterbuck in this country, and Broussais in France, each of whom has fixed upon a different region of the body as the seat of disorder.

The assemblage of symptoms manifested in the system from the presence of inflammation, we in general denominate the symptomatic fever—in contra-distinction to that which arises without any cognisable antecedent or local affection, in the language of Cullen "*sine morbo locali primario*," and which has hence been named idiopathic. It has been said that the word symptomatic or sympathetic, as used to point out the fever created by inflammation, is misapplied, in as far as it is doubtful whether the local disease gives rise to the constitutional disturbance, or whether the latter does not take the precedence of the former. Nay, farther, Dr. Southwood Smith refuses to apply the term fever, even qualified by the word symptomatic, to the constitutional symptoms in such cases; because he considers that there only can exist idiopathic fevers, and that to denote both by the same name is giving rise to a confusion of terms. "Febrile diseases," says he,[1] "are commonly divided into idiopathic and symptomatic, a division which is liable to the fundamental objection that the diseases included in the second section are not fevers, but inflammations. There are no fevers but idiopathic fevers." He adds, that the order in which the symptoms appear, and even the phenomena themselves, are different; and that, therefore, "to call both by the same name can only produce confusion and misconception."

There are certainly many circumstances connected with idiopathic and symptomatic fevers, which point out a remarkable difference between them, and which cannot be denied by the boldest opposer of this doctrine; yet the difference does not seem to depend

[1] Treatise on Fever.

so much on the symptoms present in either case, as on the nature and operation of the cause from which they more immediately proceed. If there is a point in practical medicine about which we are satisfied, it is that of the frequent existence of idiopathic fever without any local complication; and we shall just allude to a few circumstances which entitle us to consider fever as arising from other causes than inflammation. First, there is the occurrence of fatal cases of fever without our being able to detect any local cause. Secondly, nervous symptoms are often present to a much greater and more alarming extent than we ever see in fever which is evidently symptomatic. Thirdly, we find in idiopathic fever a tendency to a spontaneous termination, and which is rarely observed in inflammation. Fourthly, when inflammation does occur at an early period in idiopathic fever, we find that, although the local affection has been subdued, the febrile symptoms continue, and pursue a regular and determinate course. Fifthly, in cases where inflammation has preceded the febrile attack, or where the latter has been a consequence merely of the former, we observe that, on the removal of the local cause, the fever speedily subsides. Sixthly, idiopathic fever often exists for a length of time without any local disturbance being manifested, when, all at once towards its decline, from some accidental circumstance, as exposure to cold or the imprudent exhibition of stimulating food or drink, inflammation will arise in some local part, and the previous symptoms then undergo a corresponding change. Seventhly, the effect of the "*juvantia* and *lædentia*" shows a remarkable and striking difference between idiopathic and symptomatic fevers. We witness, for example, the great efficacy of venesection, and the injurious effects of stimuli in active local inflammation with fever; and that, on the other hand, whenever the coexistence of inflammation with idiopathic fever demands the use of the lancet, one-half the evacuation will be sufficient to subdue it. Moreover, in common fever, the most decided benefit is often derived from wine and other stimulants, even where inflammatory action is going on; and, in some cases, it appears that the only indication we have it in our power to fulfil is to prevent the pulse from sinking.

For these reasons we conceive ourselves warranted in assuming the doctrine that idiopathic fever does exist; and, farther, that it may begin and terminate without any local affection being perceptible either at the commencement, or during any part, of its course. Neither do we refuse to apply the term fever, qualified by the word symptomatic, to the effects produced by inflammation in the constitution. There cannot be a doubt that these effects are febrile, and the order of succession plainly points out that they are symptomatic of the local disease. In all such cases we observe that, while the primary cause remains, the fever continues; when increased, abated, or removed, the latter undergoes a corresponding change; and, in short, without the one we are justified in saying that the other would not have been present. The argument of Dr. South-

wood Smith has never, so far we know, been supported by any respectable authority; it is contrary to every species of evidence we possess, and a refinement, at least, which the present state of medical science will not permit us to appreciate.

The symptomatic fever arising from local inflammation, particularly that modification of it caused by local injuries, has often been described under the title of irritative—a phrase still more used when the constitutional symptoms are of a typhoid kind. The term irritative, as applied to any variety of febrile disease, is objectionable, because it does not point out the leading condition of the system, as to whether the reaction is of the sthenic or asthenic order, and because it likewise involves an hypothesis respecting the origin of the symptoms; when meant also to express the constitutional sympathy occasioned by local injuries, it is equally objectionable, for, of whatever character that may be, whether inflammatory or typhoid, or a combination of both, it is nothing more or less than febrile disturbance. We do not mean these observations to apply to the phrase irritation used in a local sense, but only when meant to signify any species or type of fever. For instance, a decayed tooth creating ulcers about the cheeks, lips, or throat, or worms in the alimentary canal producing violent itching at the nose and grinding of the teeth during sleep, are examples of irritation arising from causes which act locally, and are incapable or rather not sufficiently great to excite the phenomena of fever; but these are very different from the serious effects set up in the constitution, and which require and ought to be more explicitly defined.

Mr. Hunter first made use of the terms universal and partial sympathy to express what we have considered under the head of symptomatic fever and local irritation. By the universal sympathy is meant, when the whole constitution sympathises with some sensation or action of a part; by the partial, on the other hand, when one or more parts sympathise with some local sensation or action. These two states, the universal and partial sympathy, may be easily distinguished from each other by the circumstance, that the former is an affection of the whole constitution, while the latter is merely the sympathising of one local part with another. Partial sympathy was further divided by Mr. Hunter into three different kinds—the remote, the contiguous, and the continuous. The remote sympathy is, where there appears no visible connection of parts to account for the peculiar effects which result; as a decayed tooth creating an ulcer in an opposite part of the face, or the pain felt at the right shoulder and down the arm in inflammation of the liver. The contiguous sympathy has been defined as that arising from the contact of separate parts; thus those which are internal often sympathise with those which are external, as the brain with the scalp, the testicles with the scrotum, &c. The continuous partial sympathy is generally more extensive than either of the others; here there is no interruption of texture, and the morbid action extends from a point or centre to any distance beyond. The

spreading of inflammation along the surface of membranes or the skin in erysipelas, affords the best illustration of this form of sympathetic disease. Such was Mr. Hunter's arrangement of the phenomena of sympathy, to which many objections might now be offered. In the present state of our physiological knowledge, we can have little conception of this important process; it extends to the condition of the mind as well as to that of the body, and involves some of the most mysterious laws of the nervous system.

The symptomatic fever from local inflammation varies, according to the intensity of the disease, its seat, and the habits or idiosyncracy of the patient. It accords with what we should naturally expect—that the more severe the inflammatory action, the more intimately will the constitution sympathise with the part which is suffering. There is a reciprocity of action existing among all the functions of the animal economy, which, though unperceived, seems necessary to maintain the balance of health; and which, when subverted or disturbed, creates, as pathology teaches us, more or less extensive sympathy, according to the degree of derangement prevailing. Should an organ be affected with acute inflammation, the local and constitutional symptoms are better marked than when the disease is of a milder character; and we accordingly find that the presence or absence, or degree of disturbance in the general system, is a diagnostic mark of the intensity of the morbid action. We uniformly observe, however, that the constitutional symptoms bear a constant reference to the structure and importance of the part affected; and hence inflammation causes varieties in the symptomatic fever, according as it occupies different tissues. The serous, mucous, and fibrous structures give each of them origin to a different train of constitutional symptoms. When serous membranes are the seat of acute inflammation, the fever is of the inflammatory or sthenic type; when mucous membranes are affected, the fever is more of the typhoid or asthenic order; in inflammation of some fibrous tissues, as those placed around the joints, the local disease is not only peculiar, in as far as it translates itself from one part to another, but the symptomatic fever is remarkable for being often accompanied by diaphoresis throughout. The remarkable fact of inflammation causing different varieties of constitutional disturbance has never been satisfactorily explained; and it is best for us to express our ignorance, by referring it to an original law in our constitution.

From the regularity with which these circumstances are observed, a great dissimilarity in the constitutional effects prevails, when inflammation is seated in a containing and in a contained part. Thus, the symptoms are different when the serous covering and the proper tissue of the liver are affected. When the membranes of the brain are inflamed, the symptomatic fever runs high, the pulse is quick and bounding, there is a distressing sense of pain, and generally wild delirium. On the other hand, when the sub-

stance of the organ is the seat of disorder, the patient complains of a sense of weight rather than actual pain; he often appears overcome, feels drowsy and inclined much to sleep, and, in very young subjects, there is a conspicuous falling down or half closed state of the upper eyelid. Nay, more, we often observe that there is a considerable discrepancy in the constitutional symptoms when inflammation attacks different portions of the same organ. This is remarkably the case with respect to the brain, where the pulse will, in some cases, fall below the natural standard. This subject has been well illustrated by Dr. Abercombie,[1] with all that perspicuity, candour, and adherence to truth, which become so distinguished a medical philosopher; and to his writings, therefore, we may refer for further illustration.

Mr. Hunter was inclined to attribute the remarkable diversity in the constitutional effects of local inflammation to the connection of the viscera with the stomach. "All the parts," says he, "that may be called vital, do not produce the same effects upon the constitution; and the difference seems to arise from the difference in their connections with the stomach. It is to be observed that vital parts may be divided into two, one of which is in itself immediately connected with life, as the stomach—the other, where life only depends upon its action and use; the heart, lungs, and brain are to be considered in this last light, therefore they have a considerable sympathising affection with the stomach, the symptoms are rather depressing, the pulse is quick and small, and the blood is not pushed into the smaller vessels." In the present day we are rather disposed to reverse the statement made by Mr. Hunter respecting the organs on which life more immediately depends, and to consider the stomach as only indirectly contributing to its support; neither does it appear, in many cases where the constitutional disturbance is unusually severe, that the part has any particular sympathy with this viscus. In those cases where the local morbid action does not of itself induce death, by causing change of structure or some other dangerous product as effusion, and where the constitutional affection seems alone to have brought about the fatal event, we are inclined to attribute the extinction of life to the depressing influence exerted on the nervous system; and it is farther remarkable that, where this peculiar termination to inflammation is found, the parts are chiefly supplied with branches from the sympathetic ganglions. When inflammation has commenced in any structure not very essential to life, but with such violence as to affect the actions of life, the symptomatic inflammatory fever, and not the typhoid, is usually present; the great depression of the vital powers does not so rapidly follow; the actions of the part, as Mr. Hunter expresses it, are roused; and the effects on the constitution are not such as to impede the functions of the vital organs.

The varieties in the symtomatic fever, arising in different habits,

[1] On the Diseases of the Brain and Spinal Cord.

cannot be accurately ascertained; because it would be necessary, first, to have a precise knowledge of the extent and severity of the local mischief—circumstances which can never be compared but in a very general way. We may state, however, that the fever does not vary so much in type as in degree. Thus, inflammation of a mucous membrane in any kind of subject is accompanied by asthenic symptoms, that of a serous by sthenic; although the amount of sympathy manifested in the constitution will vary in different cases. Acute inflammation, generally speaking, proves more severe, runs its course sooner, and requires more vigorous means to subdue it in sanguineous or plethoric individuals than in those of an opposite habit. Yet, in enfeebled subjects, as those recovering from protracted fever, we often find that inflammation, brought on by imprudent exposure to cold, proves exceedingly obstinate to remove; and in fact these cases, from the fear of employing sufficiently active measures, and the tendency which the disease has to terminate in effusion, prove, without exception, the most hazardous we meet with. In conclusion, we may infer, from what we have laid down respecting the modifications arising in the constitution from local inflammation, that, where the disease does not impede the functions of any vital organ, we have, in such a case, most to fear from the severity, extent, and mode of termination of the morbid action itself. On the other hand, it may be remarked that, although much danger is to be apprehended from the local effects of inflammation when occurring in organs immediately concerned in the production of life, we have, in such cases also, to encounter the hazard from the constitutional disturbance arising out of the depressing influence exerted on the nervous system.

But, to the surgeon, by far the most important and interesting modifications of the symptomatic fever from inflammation, are those arising after the reception of severe injuries, and to which we may very shortly direct our attention. We shall here suppose that there is no tendency to any diseased action independent of what may originate in consequence of the accident, for, where a peculiar susceptibility is lurking in the constitution, we frequently observe that the parts take on a similar mode of action. Thus, in a person of scrofulous habit, an injury to a joint may ultimately lead to serious mischief, and from a cause which at first seemed trifling; or, in a female in rather advanced life, and where hereditary predisposition exists, a blow on the breast will be followed by a schirrhous tumour. These, however, are to be reckoned rather among the remote effects of accidents; and it is only to the immediate symptoms arising in the system that we wish at present to confine our observations. It is, perhaps, a trite remark, but it is equally true as trite, that no two individuals are possessed of constitutions exactly similar, and that, therefore, remarkable differences will be exhibited when they become the subjects of disease. When, accordingly, a local injury has been such as to give rise to symptomatic fever, we observe that the phenomena are varied according to the

particular habit of the patient, and it is of the highest importance for the surgeon to ascertain immediately what this may be. Before proceeding to the performance of any capital operation, the first thing done is to enquire into the general health and habit of body; and, when persons become the subjects of severe accidents, the inquiry is equally essential, for, without such information, an error may be committed at the outset which is afterwards irremediable.

The different effects consequent on the infliction of severe injuries, result from the state either of the vascular or nervous systems. The influence of the blood on the vital functions is proved by the fact, that the vigour and activity of animal life depend principally on the condition of the circulating fluid; and, according to the qualities of the mass, when inflammation sets in after severe injuries, are the subsequent constitutional phenomena in a great measure regulated. The quantity of blood in the living body is continually varying, according to the waste and supply kept up in the system; the state of health is that in which the two are most nicely adjusted, for a preponderance on either side creates disease. The composition of the mass, however, exhibits the most striking changes, and those to which our observations must be confined, as we can form no estimate of the quantity of blood in circulation. In health, the crassamentum bears always a due relation to the serum, there is an abundance of fibrin and of colouring matter, coagulation takes place in the usual manner, and the upper surface of the clot is of a florid red. In such a case, the symptomatic fever arising after an injury would, in all probability, be of the inflammatory or sthenic type, commencing and going through its stages in a regular and determinate form, not marked by extreme violence unless where the local cause was inordinate, and the constitution having sufficient powers to withstand the subsequent depressing process. Where the blood becomes redundant with fibrin and colouring matter, with a great increase in the albuminous portion of the serum, and these changes coupled with a full habit of body, there is a strong tendency manifested to disease. In such a subject, the symptomatic fever, after a trifling injury, will run high, and the subsequent prostration is equally remarkable. Contrast either of these with a case where the blood, when drawn, is seen deficient in fibrin, scarcely coagulating, or, if so, the crassamentum pale and the serum preponderating; where the nervous system is in an exhausted or irritable state, and, along with the vitiated condition of all the fluids, giving origin to the most destructive and pernicious form of fever.

Again, we have instances where the nervous system seems to predominate without any deranged state of the blood, and where the cause principally operates on this system, as some of the animal poisons, or injuries in individuals of a peculiar nervous temperament. In habits of this kind, the puncture of a clean needle in the hand will be followed by inflammation of the absorbents and glands in the arm-pit, and with violent symptomatic

fever; while frequently, after the person seems well, both the local and constitutional disturbance will be renewed on any accidental derangement of the general health. There is, however, a wide difference between the resources of the constitution in this merely nervous temperament, and that where the nervous system is morbidly exhausted by excess of stimuli and an impoverished condition of the blood, although both have been included under the familiar title of irritable. The former is principally met with in females, or in those of a delicate or susceptible frame, but otherwise in good health; the latter is found in the constitution of the habitual drunkard.

As Mr. Travers[1] has remarked, "it is scarcely necessary to illustrate the influence of an irritable temperament upon the consequences of casual injury or disease. Practically, we all know it well. We say, such a person would be a bad subject for a compound fracture; and whoever has had opportunities of watching several subjects of compound fractures under treatment at one and the same time, well knows the import of this phrase, and that the greatest degree of mischief is often accompanied by the least constitutional disturbance, and for this reason is soonest and most perfectly restored. The first few hours will enable an experienced observer to determine whether the subject of a serious injury or operation will do well or otherwise. How vastly different in different individuals is the inconvenience attending such minor derangements as a boil, an enlarged gland, a whitlow, or a simple inflammation of the eye? In some, the constitution seems ignorant of the affair, and the individual pursues his ordinary avocations. In others, the whole system sympathises—the spirits are ruffled, the nights are restless, the appetite fails, the pulse acquires an undue bound, and the white tongue, the creeping chilliness, and slight erratic pains of symptomatic fever are present. It would be of the highest importance for the surgeon, were he able to tell the constitution of a patient who has met with a severe accident, and be able at once to decide what will, in all probability, be the result of it. No doubt it is of material consequence that the local injury should be well considered, and that the greater is the mischief done to the surrounding soft parts, or the more important these are to the animal economy, the more severely may we expect the constitution to suffer. Yet a good constitution will bear up and triumph over a multitude of difficulties, while a bad constitution would not be able to support itself under one half the evil."

We may illustrate the modifications in the symptomatic fever arising from injuries, by reference to a case which often occurs in practice. An individual in sound health gets a compound fracture of the leg; the bones may be protruding, and require the integuments to be divided or the saw to be applied, before they can be brought into apposition. The man is put to bed, perhaps cold and

[1] On Constitutional Irritation.

in a state of collapse from the injury; but after a few hours, the skin gets warmer, the circulation revives, and the pulse at the wrist becomes stronger. Next day there is pain in the back and loins, with general uneasiness, a full and bounding pulse, the skin hot, the urine scanty and high-coloured, headache, want of sleep, thirst, furred tongue, and, in short, all the regular symptoms of inflammatory fever. After a lapse of four or five days, during which the fever has been kept in moderation, the general symptoms begin to subside, the pain in the head and loins disappears, the secretions are restored, the tongue becomes clean, and the sleep is natural. The injured parts, the inflammation of which had brought on the constitutional disturbance, undergo a corresponding change; purulent matter is secreted, and soon after granulations arise, by which the chasm between the ends of the bones will be filled up, and a permanent union of the osseous structure ultimately attained.

It not unfrequently happens that, during the treatment, and while every thing is going on well, diarrhœa will set in, or hemorrhage ensue from the separation of a slough; and that, in the course of a single day, all our prospects of a cure seem at an end. The purulent discharge from the wound is exchanged for a thin sanious secretion, the granulations disappear, the surface acquires a dusky hue, tending to mortification, and the chasm between the ends of the bones is as great as at first. Along with these local changes, we find that the constitutional symptoms acquire a new character; the inflammatory fever is exchanged for one of a more typhoid type, the patient gets restless, the tongue is furred, the appetite gone, the pulse sinks at the wrist, and gradual exhaustion, with low delirium, succeeds. Yet, even here, the prospect is not so dark as may be anticipated. A good constitution will bear up under a combination of evils, and may have met with such a severe shock that, until the exciting cause is removed, no chance for recovery is afforded. This is a practical point, however, which the surgeon must weigh well in his own mind; the circumstances under which he is called upon to act, are of the most trying description; there is little time for delay; but the remedy useful in one case, would in another be utterly hopeless.

To show what adverse fortune a good constitution will support itself under, we shall quote an instructive case from the writings of the late Mr. Abernethy.[1] "A man between forty and fifty years of age had a compound fracture of his leg, in which a considerable portion of the tibia was knocked out, so that the upper and lower portions of the bone lay about an inch apart. As the limb could be kept steady in a proper position, hopes were entertained that the case might do well. Granulations were produced from the periosteum and whole surface of the wound, and as those which filled the space between the broken ends of the bones nearly

[1] Lectures on Surgery.

coalesced, there was every reason to suppose that the event would be favourable. At the end of seven weeks, however, the patient was seized with a violent complaint in his bowels, when the granulations disappeared, leaving the chasm between the ends of the bones as great as at first. In his now reduced state of health, the granulations did not grow again, or they reappeared but in a trivial degree; and as his health declined, the wound fell into the state which has already been described, and produced irritative fever. The wound then sloughed, and the anterior tibial artery was laid open, in consequence of which he suddenly lost so much blood that he fainted and seemed dead, for nothing that was tried could arouse him; and in this state I amputated his thigh, tied the principal vessels, and closed the stump. He was laid in bed, still completely unconscious. Volatile salts were applied to his nose, and the warming-pan was passed repeatedly over his body; at length he gave signs of returning sensation, the circulation was renewed, and warmth was restored. He was, of course, extremely reduced; but the same kind of febrile excitement with delirium still continued for three days, when it sudsided. The wound, when opened, was found flabby, and for some time produced little or no discharge; the soft parts retracted, and, although there had at first been a superfluous quantity both of muscle and integument, the bone protruded; eventually, however, the man did well." In some cases, the patient will rally, and the limb may be preserved; while in others, the resources of the system are exhausted, and, unless the local source of disorder be removed, there is no prospect of survival. It thus happens that, in a compound fracture wherin there was at first a chance of saving the limb, amputation becomes afterwards necessary—the constitution being incapable of supporting the actions essential to recovery. Such may be the progress of a serious injury in a person of a sound habit of body.

There are two principal morbid varieties of constitution in which local injuries produce peculiar and extraordinary effects. The one is that of general plethora, attributable to over-repletion of the vascular system; the other arises from an impoverished state of the blood, coupled, in the worst species of cases, with a disturbed condition of the nervous system. We shall only select the extremes of these for examples, as it would be impossible to detail, within a brief space, the various combinations to be met with. Fortunately, such cases are easily recognised at the bedside, and their treatment must be regulated according to existing symptoms.

A plethoric state of the system, amounting to what, in common language, is termed a full or bloated habit, is engendered by over-repletion or defective secretion, and not unfrequently by a combination of both. There is a greater tendency to make blood in some individuals than in others, and in the same individual at one time than at another: when this has gone beyond a certain point, it ceases to be healthy, and brings the subject of it to the very brink of disease. When blood is drawn, it exhibits the buffy coat on

coagulation; the fibrin is increased in quantity, and likewise the animal matter contained in the serum. Such a state is far from that of health; there is a want of that tone which characterises the sound condition of the body; the muscular fibre, instead of being firm and rigid, is soft and loose; and the secretions are generally deranged. In the expressive language of Mr. Hunter, the individual is living above par. In disease, the most violent action is set up; but there is an incapability of supporting it, and exhaustion speedily follows. The full-bloated habit is principally confined to those who live high and take little exercise, or those who eat freely and use large quantities of malt liquors. Such people are easily distinguished from the rest of mankind. They do not eat and drink for the sake of living, but they live for the sake of eating and drinking. To use the language of Prior, who has not inaptly described them,

> "They eat, and drink, and sleep; what then?
> Why, sleep, and drink, and eat again."

Not unfrequently, a trifling circumstance, such as would hardly have been perceived by a good constitution, is followed by the most serious consequences. Thus we have seen a fretful chilblain over the heel giving rise to inflammation, spreading in patches throughout the limb, and ending in a succession of large sloughs, under which the patient speedily sank; or a splinter of wood driven under the nail of a finger, creating such violent constitutional disturbance as to terminate fatally within a few days from the commencement. Nothing, therefore, is more characteristic of this habit than the amount of reaction, compared with the nature of the cause which may have induced it.

When a serious injury, as a compound fracture, occurs in this state of the constitution, symptomatic fever of the most violent and dangerous description is set up. There is at first great excitement of the whole vascular system, followed by a corresponding degree of depression; there is a disposition to action, without sufficient strength to maintain it; and, from the rapidity with which these changes succeed each other, the struggle is often short. Within forty-eight hours after the receipt of the injury, inflammatory fever of the most vehement kind is present. The tongue is dry, brown, and deeply furred; the urine high coloured, and its secretion nearly suspended; the skin burning hot, and the face flushed; the thirst incessant; headache throughout the day, followed by furious delirium requiring restraint throughout the night; the respiration hurried, and the breath offensive; the belly costive, and the evacuations of the colour and consistence of pitch; great general restlessness, with incessant groaning; and a full and labouring pulse. If immediate relief be not afforded, by bold and decided treatment, the scene quickly changes into one of an opposite character, marked by apparent debility and failure of the powers of life. The wild delirium settles into one more calm, and of longer

duration; the face becomes livid; the lips and teeth covered with a black sordes; the breathing stertorous; the skin bedewed with a clammy sweat; the extremities cold; the pulse at the wrist flagging, irregular or intermitting, rapid and feeble; profound coma winds up the tragedy, and the individual dies oppressed. On dissection, we have found the veins of the brain distended; effusion of serum between different parts of the arachnoid covering, and into the ventricles; the substance of the hemispheres slightly softened, and presenting numerous red points when cut across; in some cases the lungs gorged with blood, and an increased quantity of fluid contained within the pleura. Nothing, at the commencement, will suffice but free, general, and local depletion, with purgatives; and we have, by these means, known consciousness restored, after an unfavourable prognosis had been passed.

But by far the worst kind of constitution we meet with, and one where the least hope of saving life may be entertained, is that where the blood is impoverished, and the nervous system in an irritable and morbidly-exhausted state. This is found in the drunkard who consumes large quantities of ardent spirits, gets no regular meals, or whose food is not of the most nutritious description. This sort of individual, as Mr. Hunter would have expressed it, is far below par; he possesses no resources within himself, and sinks under the slightest affliction. If a rigid system of abstinence is pursued, the constitution is speedily unhinged; and we have seen, from this cause, a simple fracture of the leg, or a trifling rheumatic affection of the joints, prove fatal. Those who are great eaters or great drinkers are equally bad subjects for accidents or operations; and it is almost impossible to declare which is the more dangerous of the two.

When a person, much addicted to the use of ardent spirits, has received such a severe injury as a compound fracture, we often observe that the symptoms from the commencement are of a peculiar cast. The state of collapse immediately following the receipt of the mischief is long in subsiding; there is little tendency to reaction; and the nervous system seems exhausted by the shock which it has experienced. Next day, low typhoid fever is present; there is slight heat of skin, with general restlessness; the stomach is irritable, and frequently much uneasiness is felt about the præcordia; the tongue becomes dry, and covered with a deep brown fur; the pulse is rapid and feeble; the patient has been in a state of wakefulness throughout the night, or slept disturbedly for short intervals; and the intellect is slightly confused. These symptoms continue to progress. Hour after hour the prostration of strength increases; the hand gets tremulous, and the mind wanders from object to object. If we can engage the patient's attention, he will, perhaps, answer us correctly, then break out into some foolish exclamation, or address some strange objects which he imagines are dancing around him. Ultimately, the whole frame is agitated; the tongue cannot be protruded; the pulse dies away at the wrist;

the restlessness ceases; and a low muttering delirium, ending in coma, succeeds. During the prevalence of these typhoid symptoms, there is generally a determination of blood towards the head; the face is flushed, and the temporal arteries are beating violently; and, when the brain is examined after death, marks of subacute inflammatory action, with increased effusion, are found.

In the worst variety of cases, the dismal train of symptoms now described arises and terminates within two or three days from the receipt of the injury; in others, some little time will elapse before the constitution breaks up and low fever sets in. The one is unmanageable from the commencement; the other has, perhaps, become so from the want of proper precautions. Both, however, form a complete contrast to the over-plethoric habit. In the former, there is an absolute want of power, a direct debility; the blood is deficient in fibrin, and the nervous system is exhausted. When this class of persons are the unfortunate subjects of accidents, depleting measures, if necessary, must be sparingly employed; for we have seen general bleeding followed by the most fearful effects. The nervous system seems, in the first place, to suffer from its customary stimuli being abruptly withdrawn; and hence, while local means are used to subdue any dangerous inflammatory action that may be going on within the cranium, opiates and stimulants must be given, in divided doses, to support the action of the heart. Few cases are better calculated to draw forth the skill and judgment of the medical practitioner than these, although it must be confessed that they but too seldom reach a favourable termination.

SECTION V.

VARIETIES IN THE PROCESS OF INFLAMMATION.

These may be considered under two heads—either as appertaining to the morbid action itself; or, as connected with some specific cause residing within the constitution. Inflammation is, therefore, common or specific. Of the former, the most ancient division was into phlegmonous and erysipelatous, or healthy and unhealthy; a division which, even in the present day, is not entirely laid aside. Phlegmon (from Φλεγω, to burn,) was deemed healthy, because it showed little disposition to spread; and when matter was formed, the fluid was found to be confined within a sac or cyst. Erysipelas, again, was reckoned unhealthy, because no limitation could be placed to its spreading over all the body; and when suppuration followed, the matter was diffused throughout the cellular tissue of the part. To this source are to be traced the erroneous notions yet entertained by many respecting erysipelas, and also the singular idea, that inflammation of the skin differs from all others, not

merely in its seat, but in its nature. Whatever opinions may be held respecting phlegmon and erysipelas, these terms express neither the duration or intensity of the inflammation; we now consider the former as occurring in the cellular membrane which constitutes the basis of the different organs and textures throughout the body, while the latter is restricted to the skin and subjacent tissues. On the grounds, therefore, of involving an hypothesis as to the nature of the morbid action—which seems at variance with the truth, and resides only in the imagination; and as denoting neither the degree or length of time during which the affection has lasted—the division of inflammation into phlegmonous and erysipelatous, or healthy and unhealthy, should be rejected.

The process of inflammation, in some cases, is violent, and its progress equally rapid; in others, it is languid, and remains with occasional remissions and exacerbations for any length of time; hence it has been described as either acute or chronic. No fixed period has been given by which we may determine when inflammation comes under the one or the other of these heads. When the disease begins and terminates within three or four days, we say that it has been acute; when continuing for several days, it is reckoned of a chronic character. These terms are, however, employed not merely to signify the duration of inflammation, but likewise to denote the difference in its symptoms and progress. When we make mention of an acute inflammation, we mean one which passes rapidly through its different stages; which terminates speedily in some manner or other; and in which both the local and constitutional symptoms have been severe. When, on the other hand, we speak of a chronic inflammation, we understand one which has remained some length of time, perhaps, unchanged in its character; in which neither the general nor local disturbance is well marked, but which leads, ultimately, to permanent disorganisation of the affected part. The phrase acute only expresses the nature or character of the process itself, without any reference to time; while that of chronic, (from χρόνος, time,) without implying the kind of morbid action present, signifies merely that it must have been of some duration. In the proper sense, indeed, in which these terms should be used, practitioners greatly err; for we are in the daily habit of employing them to designate more the degree than the duration of inflammation. In no two individual cases, however, is the severity of the local and constitutional symptoms alike. Inflammation does not consist of two states only, an acute and a chronic; between these extremes many shades of difference might be recognised; and, accordingly, to express some of these modifications, the phrase subacute has been introduced. It is quite impossible to distinguish by mere names, all the various degrees of a morbid action, which, at one time, assumes an indolent, and, at another, a most violent character, and of the intensity of which, the best criterion is the general and local disturbance.

In order to avoid any misconception arising from the use of the

terms acute and chronic, those of active and passive have been introduced; but the latter, in the sense in which they are ordinarily employed, are nearly equally objectionable. In many cases which are considered chronic, the inflammatory action is by no means of a passive or indolent character, but, on the contrary, of an active nature, and requiring vigorous means for its removal; while, in others of an apparently active description, it is in reality passive, and does not admit of the same resolute treatment. We have frequent examples of the former in rheumatism, where, although the disease may have continued so long as to merit the name of chronic, it is, nevertheless, of an active character, and may demand the use of the lancet for its removal; while instances of the latter are occasionally furnished in subjects debilitated from fever or other causes, where inflammation may be acute as regards its duration, but has more of a passive than an active disposition. True, in the majority of cases, the terms acute and chronic correspond exactly with those of active and passive—most acute inflammations being of an active kind, while chronic partake of the passive order; nevertheless, the exceptions to the rule are so numerous, that, where we wish to express the degree as well as the duration of the disease, we would, on some occasions, require to make use of both phrases at the same time.

It is a matter of doubt, also, how far we are justified in speaking of inflammation being altogether passive, for more or less action is continually going forward, and the morbid parts are not observed to be in that state of rest which the term passive would imply. In organs where this form of inflammation has been long present, changes in their bulk and structure have been gradually taking place—showing that the inflamed vessels are not in a state of total inactivity. In acute inflammation, the action of the vessels is said to be increased, while, in the chronic form, it is diminished; and this, we are generally told, constitutes the principal difference between these opposite varieties. It was one of the conclusions at which Dr. Thomson arrived, when watching the process of inflammation in the web of the frog's foot, and it has been referred to as an ultimate fact in pathology. This is one among many points in practical medicine, which, from having remained undisturbed, has been invariably assumed as true. We do not mean to assert that, in numerous cases of chronic and lingering inflammation, the vessels are not in a state of debility and relaxation, because both the appearances they present and the effects of certain remedies proclaim them to be so; but we mean to be understood that this does not hold true in every instance. Neither does acute inflammation alone consist, as such notions would seem to inculcate, of increased action throughout; for soon after it is established, and if its phenomena are at all well marked, we find that the minute vessels are paralysed, and the fluid is stagnant within them. In short, in chronic inflammation we have no reason to believe that the capillaries are in such a state of debility, or the circulation through them

so much interrupted, as towards the termination of the disease in parts which have suffered more severely; while, again, the fact of the chronic form being often the result of that which has been acute, and of the latter arising at any time out of the former—the facility of change from the one to the other being very great—inclines us to consider them as the same process, differing rather in intensity than in any supposed increase of activity or debility in the vessels themselves.

We need not, however, have recourse to abstruse reasoning, in order to distinguish between acute and chronic inflammation; neither would any minute changes in the vessels at the seat of disease enable us, without more obvious means, to establish the difference. Acute inflammation may be generally described as either advancing or declining; seldom or never stationary; running its course quickly; ending in resolution or mortification, or forming some new product. It is especially recognised by the severity of its local and constitutional symptoms; the part either speedily recovers its former condition, or experiences a serious alteration in structure, and the symptomatic fever undergoes a corresponding change. Although more violent in its character, acute inflammation is, nevertheless, far more tractable and readily removed than that which assumes an indolent and determined character; neither do we observe, during the presence of the former, that increase of size and change in the conformation of organs, so conspicuously seen during the maintenance of the latter. No difference is apparently manifested between parts which become the seat of disease in the way of giving rise to one variety rather than another; for, as far as we have been able to remark, acute or chronic inflammation may arise in every organised tissue in the body.

To avoid any confusion respecting congestion and inflammation, Andral has substituted for these the term *hyperæmia*, which signifies an excessive quantity of blood in a part; but this cannot remove a difficulty which consists not alone in words, and we think it better to retain those, the use of which custom has long sanctioned. Inflammation has also been described as tonic or atonic—sthenic or asthenic—latent—sympathetic—continued, as in that we daily meet with—intermittent, as in some forms of ophthalmia—and remittent, as said to occur occasionally in children; but, with the exception of those which are latent and sympathetic, the rest have either no foundation in nature, or occur so seldom as to be unworthy of any separate notice. Inflammation has, farther, been supposed to vary according to some peculiar states of the constitution, as the scrofulous, the syphilitic, the gouty, and the rheumatic. In such cases, the disease has been represented as of a specific character, although the difference is not presumed to depend so much on the process itself, as on the habit of body in which it appears. In the scrofulous subject, inflammatory action is often modified, and gives rise to some peculiar changes, which, during our progress, we mean to consider; in others, as the syphilitic, the

difference is only ideal, but has been the fertile source of the pernicious treatment followed out in numerous cases; the remainder do not properly come within the scope of our investigations.

SECTION VI.

TERMINATIONS OF INFLAMMATION.

We proceed briefly to detail some of the modes by which inflammation terminates. These may be described under the heads of resolution, adhesion, effusion, suppuration, ulceration, mortification, and hardening and softening of tissues. Of late, much quibbling has arisen as to the propriety of applying the phrase termination to some of the changes induced by local inflammation, and which, it is contended, ought to be regarded as so many effects or consequences. If we understand, by the word termination, that the morbid action has completely ceased, then we should use it in too extended a sense; for, with the exception of resolution and mortification, we have no other instance where the previous inflammation does not still accompany, in a less degree perhaps, the particular change which has ensued. Thus, where purulent matter is formed as a product of inflammation, we know that the latter has abated in intensity, but not yet subsided; and the same may be observed in some of the other effects which ensue. When the word termination is employed to signify that the local disease has undergone some alteration, or that some new change has taken place in the part, we apprehend that about as little error is committed as when we apply the term effect to denote the same thing; for, even in a strictly physical sense, where we make use of the latter, we often presume that the cause has ceased to operate. These objections to the word termination show a wonderful degree of ingenuity turned to little purpose; for, as few will be liable to mistake our meaning when we say that inflammation has ended in suppuration as in mortification, and we shall accordingly continue to employ the phrase in the manner now explained.

The most frequent and natural termination of inflammation is that by resolution. Here the morbid action disappears, and the healthy function of the part is sooner or later restored. With the exception of mortification, this is certainly the only other instance where all traces of morbid action are removed; and hence the reason, as we have remarked, of the other changes having been regarded as effects. The French pathologists have applied the term delitescence (from *delitesco*, to abscond) to cases in which inflammation suddenly disappears; but, in some of these, the disease has only shifted its seat from one part to another; and such instances cannot, strictly speaking, be included under the head of

resolution. Were we to understand, however, under this head, that there was a complete cessation of every morbid alteration in the solids, and a restoration of the natural powers and functions of a part, there would be many cases which would be excluded, although the inflammation had, without doubt, been subdued. Where the disease has been acute, and lasted for even a moderate time, it invariably gives rise to effusion of serum or lymph, or blood may be extravasated from some of the ruptured vessels; and when we succeed, in such cases, in putting an end to the incited action, we can scarcely say that a complete resolution has been brought about. Neither, where inflammation has been intense, do we find that the part or organ in which it has been seated regains its lost function suddenly, but in the most gradual manner, remaining, in fact, weak, and liable to a renewal of the disturbance from the slightest exciting cause. Provided, however, the effusion into the tissue of the part is trifling, it is soon removed by absorption after the undue action which led to it has ceased; nor is there any necessity, or would it be possible, at all times, to separate such cases from those wherein the inflammation has been previously subdued.

The termination by resolution is attended with different results, according to the structure which has been affected. Thus, in serous membranes, further effusion is checked, and what is already thrown out absorded; from mucous surfaces, the discharge is for a time both increased and altered in character. The changes going forward at the seat of disease during resolution, are—the diminished action of the larger arteries leading to the inflamed spot, and the gradual decline of the *vis a tergo*—the contraction of the over-distended capillaries—the propulsion or removal of the red blood from the colourless arteries into the veins—and the re-establishment of the natural circulation. While these phenomena are taking place, the local and constitutional symptoms abate; the pain, heat, and redness, disappear; the pulse falls to near the usual standard; the thirst and heat of skin subside; and the secretions are restored.

In resolution effected by natural causes, we have almost invariably observed in the mesentery of the rabbit, that the first step in the process was the cessation of the undue action of the larger arteries leading to the inflamed spot, by which the impetus of the blood's motion is lessened, and by which, also, the weakened capillaries are enabled to contract and regain their tonicity so as to carry on the circulation. A considerable interval of time elapses before the whole of the morbid phenomena subside, and the part assumes its former appearance; in some places, we observe the vessels dilated and the blood at rest; in others, the fluid is moving slowly forwards; while in others, the healthy balance seems restored. When a stimulant, as turpentine or a solution of nitrate of silver. is applied to the inflamed part, it causes a sudden contraction of the vessels and a paleness; the impetus of the larger arteries,

however, remains unchecked; and, unless the contraction is permanently kept up, the capillaries are afterwards farther weakened by the increase in the general circulation arising from the stimulus employed. The phenomena thus observed in the lower animals, correspond with what we frequently witness in the human subject from the untimely application of stimulants to parts labouring under active inflammation, and where means have not first been made use of to lessen the circulation. They likewise point out the advantage of keeping the system in a state of tranquillity after the morbid action has abated, from the facility with which, at this period, the latter may be renewed.

When two inflamed surfaces are brought together, and kept in apposition for a certain period of time, an intimate union is established between them; they are then said to adhere, and this is the meaning of the termination by adhesion. The uniting medium is lymph, which subsequently becomes organised—constituting the process by which recent incised wounds are generally healed. The fibrinous portion of the blood is thrown out more or less readily, according to the structure attacked or the necessity for its separation. Thus it is effused on the surface of a wound or a serous tissue, under a slight increase of vascular action; while adhesion of a mucous membrane is only effected by the most intense inflammation. We have already adverted to the fact that, where much lymph is thrown out during inflammation, as in some cases of serous membranes, less fibrin will be found in the blood drawn from a vein; but this, although accounting for a deficiency in the buffy coat where the local inflammation may be severe, is inadequate to explain the cause of its formation. The lymph thus effused, and constituting a living bond of union between two opposite surfaces, has been supposed to vary in its nature from that ordinarily found in the blood; the circumstance, however, to which we have just alluded, shows the intimate connection subsisting between them. Mr. Hunter has treated of these phenomena under the title of "the adhesive inflammation;" and certainly wherever such changes are going on, more or less of inflammatory action is always present.

When the exhalant vessels of a part labouring under inflammation throw out a quantity of serous fluid, and by which the local action may probably be diminished, we understand the termination by effusion to have taken place. In some cases, the exhalation has the effect of relieving completely the inflamed vessels; in others, the relief is only partial, and the incited action is still kept up. The effused fluid seldom consists of pure serum; frequently it is tinged with the colouring matter of the blood, or mixed with portions of lymph; or should the inflammation be violent, with flakes of purulent matter. We have already hinted that, in the great majority of cases, there is an exhalation of serum with a little lymph, or even an extravasation of blood; but where the effusion is trifling, and incapable of producing bad effects, we still consider

the disease to have ended in resolution. The termination by effusion principally takes place when a serous or cellular tissue is affected; and in the former, the degree of danger is to be estimated by the quantity poured out and the seat of extravasation. When effusion is the result of acute inflammation within the cranium or chest, the case is attended with the most imminent danger—the patient dying of asphyxia, commencing either at the brain or lungs; when occurring in a more chronic form in the abdomen, it gives rise to a variety of ascites; in an external part it merely creates œdema, and pain or inconvenience from over-distension.

When the local and constitutional symptoms have been severe, followed by rigours or shivering, the pain of the inflamed part gradually remitting, and fluctuation becoming perceptible, the process of suppuration has then ensued. The formation of purulent matter is frequently preceded by a throbbing pulsation at the seat of disease, and takes place with more or less facility in different structures. The previous morbid action, though lessened in severity, still remains for some time, and Mr. Hunter accordingly called it the "suppurative inflammation."

When a portion of the inflamed structure is removed by its own absorbents, and leaves an abraded surface from which a discharge is furnished, the phenomena of ulceration are then established. The removal of parts of the animal body by ulcerative absorption must be carefully distinguished from that gradual absorption of structure caused by tumours or foreign bodies making their way to the external surface; the former is accompanied by pain, heat, redness, and secretion; the latter is unattended by either. Mr. Hunter was of opinion that the ulcerative inflammation was often substituted to prevent a part from perishing; the morbid action is, however, less severe than that by which the vitality is destroyed, and seems to occur very readily in certain textures, as skin, cartilages, and mucous membranes.

When the severity of the local symptoms suddenly remits, the inflammatory fever is exchanged for one of a typhoid character, and the part observed to be passing from a livid to a dark colour, and losing its vital heat, mortification is rapidly advancing. The process has been usually divided into two stages, viz.—gangrene or incipient mortification, and sphacelus or complete death of the part; and, with the exception of resolution, it is the only other instance wherein the local inflammation completely disappears. Mortification may occur independent of inflammation, as in some peculiar states of the constitution, or from the continued application of intense cold; as a termination of this disease, it takes place more readily in some individuals, and in some textures, than in others. We forbear entering at greater length on the more ordinary effects of inflammation, as it will be necessary to revert to them at a future period.

Softening, or loss of cohesion, is a common effect of inflammation; and, in tracing the progress of the disease in different parts

of the body, we shall have occasion to mention this as of frequent occurrence. Softening is more particularly observed in some tissues or organs than in others, as in mucous or cellular membrane, and in the substance of the brain. It is generally caused by effusion of a serous fluid between the laminæ of a structure, but at other times consists in a direct loss of cohesion in the proper fibre of which a part is made up. Softening is more frequently the result of acute than chronic inflammation, and was first described in the brain and spinal marrow by the French pathologists under the title of *ramolissement*. The loss of cohesion varies in different parts, according to their structure and the diseased action present; thus, the substance of the brain may be reduced from its natural firmness to a semi-fluid mass resembling thin custard. Softening of this organ, however, may take place from other causes, as ossification of its vessels; and, in some cases where the affection is extensive, it has been doubted whether it was really the consequence of inflammatory action.

Induration is the very opposite of that to which we have alluded, and is occasionally the effect of inflammation. It more frequently arises from the chronic than the acute form of the disease, and is caused in various ways, according as the one or the other of these has been present. In acute inflammation, the induration proceeds from the effusion of lymph and a highly albuminous serum into the interstices, and between the laminæ, of the tissue; when resulting from the chronic variety, there is frequently a deposition of a new and solid product, different entirely from the natural structure of which the part is made up.

Induration commonly occurs in the cellular membrane, or in parts abounding with it, as the lungs; and in soft organs, as the brain or liver. It is sometimes accompanied with increase of the part affected, constituting *hypertrophy*; at other times, with wasting of the natural structure, giving rise to *atrophy*. Induration with hypertrophy may be caused either by a superabundant deposit of the nutritive matter which enters into the composition of the part, or by the formation of new products, as particularly seen during long-continued chronic inflammation. The degree of hardening depends principally on the nature and density of the superadded matter, as frequently little change arises in the primary texture itself. When the new deposit is such as is met with in other parts of the body, it is named an *analogous* transformation; when different from any known natural structure, a *non-analogious* transformation. From the induration caused in parts by inflammation, scirrhus has been looked upon by some as one of its terminations; but true or malignant scirrhus is a specific disease arising from hereditary disposition, and quite independent of common inflammatory action. In consequence of the new matter deposited into the cellular interstices of parts, we find their colour altered, and their bulk and weight much increased; and, in this way, chronic inflammation may give rise to a great variety of effects. In some

instances, both softening and hardening are combined, occupying different portions of the same texture. Finally, in estimating the changes arising from inflammation, it is always requisite to compare them with the consistence of the tissue in the healthy state, together with its density at various ages.

SECTION VII.

PROGRESS OF INFLAMMATION IN DIFFERENT STRUCTURES.

There is no organised structure in the animal economy in which the phenomena of inflammation may not be observed; and, in this respect, the disease differs from all others we are acquainted with. The human body is made up of different tissues, in each of which, inflammation gives rise to a peculiar train of morbid changes; and we must, therefore, be prepared to meet with various effects, according to the seat of disease. The attention of medical men was first drawn towards this subject by Dr. Carmichael Smith, in 1788;[1] and subsequently, in France, by Pinel and Bichat. It is not our intention to enter minutely into many parts of this investigation; but the great importance of studying it may be estimated from the space latterly devoted to it in the writings of many distinguished pathologists.

Cellular Membrane.—The cellular or reticular membrane, so extensively diffused throughout the body, and constituting the basis of all other parts, is the most common seat of inflammation. The cellular tissue has been generally considered as giving origin to phlegmonous or healthy inflammation; but experience teaches us that it is equally involved in an erysipelatous or unhealthy action. In acute inflammation the reticular membrane loses its elasticity, and is easily broken down; the cells are filled with a gelatinous reddish-coloured fluid, an in some places with pure blood; the minute vessels are distended, and a yellow serum is effused around the inflamed spot. The morbid action may terminate in resolution, effusion of lymph or serum, formation of matter, or mortification and sloughing. Primary ulceration never occurs as an effect of inflammation in the cellular tissue.

When acute inflammation originates in the cellular membrane, and ends in suppuration, the fibres become soft and loose; the pus is confined within a cyst formed of the condensed cells, and varies in its character according to the seat of disease; but when the inflammatory action spreads from the skin to the reticular texture beneath, adhesions are often imperfectly formed, and the purulent fluid undulates through the membranous cells. The different effects thus observed from suppuration, in the same texture, are principally attributable to the circumstances under which it takes place. In

[1] London Medical Communications. Vol. II.

the one case. the inflammation is circumscribed, lymph is copiously thrown out so as to form a boundary to the abscess, and the accompanying fever is usually of a sthenic type; in the other, the morbid action is more extensive, often violent, and occurring in an unhealthy habit, while the symptomatic fever is generally of a typhoid description. We have examples of the death or sloughing of the cellular tissue in erysipelas, bile, carbuncle, hospital gangrene, and malignant pustule; in some of these, the membranous fibres separate, and come away in masses like wet tow; they originate either in a depraved state of the general health, or from inoculation with an animal poison, and are accompanied invariably with low typhoid fever.

When the adipose tissue is inflamed, the fat is broken down and mixed with lymph, serum, or extravasated blood; and, should suppuration ensue, the fat is absorbed, and purulent matter deposited in the cells it previously occupied. In parts abounding with adipose substance, and where the pus is deep-seated, as in the neighbourhood of the rectum, the fluid is generally fœtid, and the constitutional symptoms of a strongly-marked typhoid character. When chronic inflammation attacks the cellular tissue, the fibres are thickened, and the cells filled with an albumino-gelatinous fluid; the part thus acquires an increased hardness and density, loses its natural elasticity, and, on being cut into, presents a grayish or spotted red appearance. Such, at least, is the condition of the cellular membrane in what is named skin-bound disease.

Arteries.—The arterial tissue is not so readily inflamed as has been supposed. The subject has only of late years attracted notice; but there is reason to think that, like every other new doctrine in medical science, a degree of importance has been attached to it of which it is unworthy. Although the capillary branches are the proper seat of inflammation, the larger arteries do not often exhibit the same phenomena. Both acute and chronic inflammation have been described as occurring in the arterial texture. The internal membrane which lines the vessels resembles closely, in structure as well as function, the serous tissues; and, when inflamed, undergoes changes analogous to what is found in these. The adhesive form is that generally seen in the tunics of arteries, from whatever cause the inflammation proceeds; there is increased vascularity at the spot affected; the *vasa vasorum* are more numerous and distinct; and the middle coat is softened. On more minute examination, the smooth polished appearance of the inner tunic is observed to be lost, and the membrane itself thrown into folds.

We must be careful not to mistake colouring of the internal tunic from imbibition after death, for the effects of disease; and we are persuaded that many errors have arisen in this way. Mere redness of the lining membrane of an artery is no proof that inflammation had existed, because this takes place frequently from transudation, and particularly, as we stated, in cases of sudden death, or where there had been any obstacle to the free circulation of the blood. When the redness occurs in numerous places throughout the

vessels, unaccompanied by any other signs of disease, we may rest assured that it is merely the effect of transudation; when, on the contrary, it is confined to one part of the vessel, along with it we observe the nutritious arteries to be enlarged, the surface of the inner membrane opaque and easily scraped off, with tumefaction and effusion of lymph, we are warranted in concluding that inflammatory action had been present.

The most common effect of acute inflammation in arteries is softening of their tunics, effusion of lymph in the connecting tissue between them, and on the surface of the internal membrane—the latter likewise loses its transparency and is thrown into folds. When the local action is severe, purulent matter may be effused between the coats or secreted from the inner membrane, and mingle with the circulating fluid; the vessel is increased in bulk from the infiltration, and so softened that laceration readily takes place. Portions of the artery will sometimes die and slough away, and then hemorrhage ensues; more commonly, however, the inflammation gives rise to effusion of lymph, by which the tunics are agglutinated, and the canal of the vessel rendered impervious. Whatever some modern pathologists may say to the contrary, acute arteritis is a rare affection; it takes place to a certain extent when an artery is wounded or surrounded with a tight ligature, but its spontaneous occurrence in a large trunk is seldom seen; and, when occurring in those which are deep-seated, there are no signs by which we could detect it during life. The idea of acute inflammation occurring universally throughout the arterial system is so absurd as scarcely to merit notice; and we are confident that, in many other instances, the effects produced by transudation have been mistaken for disease.

Chronic arteritis is said to be of more frequent occurrence than acute. It is principally confined to the larger vessels, causing a deep brown tint of their tissue, with softening and rugosity of the lining membrane. Long-continued chronic inflammation gives rise to ulceration of the serous tunic, which may extend through the whole thickness of the artery, and induce fatal hemorrhage; we have a preparation where a minute opening was thus formed in the aorta within the pericardium, and led to instant dissolution, by the filling of this cyst with blood. Ulceration not unfrequently takes place in the lining membrane of the heart, as Laennec described.

Nothing is more common in the larger arteries than to find morbid deposits of a calcareous, cartilaginous, or osseous substance, within their tunics; occurring in patches of greater or less extent, and occasionally extending for some distance throughout the entire circle of the vessel. This degeneration has been considered as the effect of chronic inflammation, and chiefly for the reason that dark brown spots are observed in the neighbourhood. They often occur in parts of the vessel distant from each other, and unconnected with any other morbid appearance. They seem rather new deposits into the substance of the middle tunic, or between it and the inner

membrane—for the latter may be scraped off entire; the same phenomena take place in other tissues; and hence they may, with as great probability, be the cause, as the consequence, of chronic inflammation. In whatever way they originate, when occurring partially in an artery they are a frequent source of aneurism; the diseased portion of the vessel is incapable of yielding with the neighbouring sound one to the impulse of the circulation, and, being once rent, the blood insinuates itself through the opening, and distends the outer tunic into an elastic swelling. They are likewise of great importance to the surgeon in considering the cure of this disease by operation.

Veins.—The lining membrane of the veins is very susceptible of inflammation, and the disease gives rise to the most fatal effects. Phlebitis never originates spontaneously, but is the result of accidents or operations, or some other cause in which the vessels are directly concerned. It thus follows venesection at the bend of the arm, or the division or tying of veins after amputation, or for varix; it is a frequent though unsuspected cause of death after lithotomy; it gives origin to one of the varieties of puerperal fever; and produces also *phlegmasia dolens*, the pathology of which was so long misunderstood. We have seldom known it succeed after severe injuries or operations in individuals of sound constitution; but, in an irritable or exhausted subject, especially where the suppuration is profuse, it not unfrequently supervenes. The first distinct account of inflammation in veins was published by Mr. Hunter in 1793; but notwithstanding, the disease, till of late years, attracted little notice.

Acute phlebitis usually commences within a few days from the application of the local cause; and in some cases proceeds with such rapidity as to resemble the introduction of a specific poison within the body, over which medical treatment has no control. At other times, a wound will be in a healing condition and no unusual suppuration present, and the disease will advance more slowly but steadily to a fatal termination. In deep-seated veins, the local signs are often so obscure as to escape observation during life, and the constitutional effects have been attributed to another source; hence phlebitis ought always to be suspected, where violent typhoid symptoms ensue after wounds or operations without sufficient local cause.

When the vein is examined after death, the lining membrane is found intensely red, and, along with the outer tunic, so much thickened as to make the vessel feel like a hard impervious cord, and resembles an artery in appearance. The tissue in which the vein is imbedded is likewise unusually vascular, being infiltrated with a reddish serum, much softened, and in some cases of a dark-brown or black colour. The canal of the vein is filled with lymph, either loose or adhering to the inner tunic, and obstructing the passage; there are deposits, also, of purulent matter, sometimes so copious as to distend the vessel. The pus is frequently confined

by the lymph forming boundaries to it both above and below, and in which case a chain of little abscesses occurs throughout the course of the vein. The inflammation may be limited to a small portion of the tunics, or spread upwards and downwards throughout a great extent of the vessel. In venesection at the bend of the elbow, or in wounds of the femoral vein at the groin, the disease may proceed towards the fingers or toes, or in the opposite direction towards the heart, or, what is more common, in both ways at the same time.

Mr. Hunter has stated that death takes place as soon as the inflammation reaches the lining membrane of the auricle, but in very few instances does the local mischief extend so far; while the fact —of the disease sometimes taking an opposite direction, and of its proving fatal where only a few inches of a vein are inflamed—renders it obvious that some other cause of dissolution must prevail. Mr. Arnott[1] has remarked that the point at which the inflammation of a vein ceases is either where the vessel sends off or is joined by another branch. Thus, where a trunk is concerned, the boundary is the entrance of a branch; and where a branch is affected, the boundary is its junction with a trunk. He does not mean to infer from this, that the spreading of the inflammation must necessarily be arrested, but merely to remark that, where it does cease, there is commonly found another current of blood. His observations have led him to the conclusion that the fatal indisposition is to be traced to the entrance of pus or some other product into the circulation; but this, although it may simply induce death in some cases, fails in doing so in others, and the bad effects, as we shall immediately see, are often referable to the complications of other organs. The chance of purulent matter being mingled with the blood is much greater where large than small veins about a wound are affected.

The constitutional effects resulting from inflammation of veins vary according to the extent of the local disease, but may all be considered as of the typhoid or adynamic order. At the elbow joint, while the affection is limited, the febrile disorder is moderate; while the local symptoms are well-marked by the pain in the wound and along the track of the vessel, which is hard and knotted, and by the swelling of the limb. When the inflammation is greater, and purulent matter has formed, the secondary effects in the constitution are materially altered; and where the local changes are concealed from view as in some of the deep-seated veins, and there is nothing to indicate the disease externally, the febrile disorder has been mistaken for malignant typhus alone. There is at first great anxiety of countenance and depression of spirits, an irritable state of the stomach, and a distressing feeling of general uneasiness, without the patient being able to refer his symptoms to any particular source. To these succeed heat of skin, and thirst, with a pulse ranging from 100 to 160 in the minute, and a hurried and anxious

[1] Medico-Chirurgical Transactions. Vol. XV.

respiration. The sensorial powers are soon affected; there is incoherence, with either low muttering, stupor, or occasional attacks of violent delirium, followed by exhaustion and a partial return to consciousness. Before death, and as the disease progresses, all these symptoms are fearfully increased; not unfrequently the skin turns yellow, leading to the belief that the patient is jaundiced; the features sink, the pulse gets rapid and thready, the tongue is of a dark brown, and the teeth are covered with a black sordes, tremours of the muscles of the face and extremities follow, and the patient lapses into a state of fatal coma. Acute phlebitis, although a most dangerous, is not invariably a fatal disease; when recovery does take place, we find that the canal of the vein is rendered impervious by the effused lymph, and that the vessel subsequently degenerates into a round ligamentous-looking substance.

In many cases of phlebitis, secondary abscesses occur in distant parts of the body. The late Mr. Rose of St George's Hospital published a paper on the purulent depositions found in some of the viscera after severe injuries or operations, without, however, being able to explain correctly from what source they proceeded. Such cases have been described under the title of sympathetic inflammation; but they are all referable to inflammation of the veins, and the admixture of purulent matter with the blood in circulation. In some, the eyes are the seat of purulent ophthalmia, the lids become red and swollen, pus is effused into the interior of the globe, and the cornea is rendered opaque and finally bursts. In others, the fleshy substance of the thigh, or the joints of the knee or elbow, are the seat of mischief; and, on laying open the cavities of the latter, we find a mixture of pus and other fluids, and the cartilages stript from the articulating ends of the bones. More frequently some internal organ, as the brain, lungs, liver, or spleen, is softened and in a state of partial gangrene, and in some part of its substance is found a cavity filled with fetid purulent matter. Sometimes the functions of organs in which pus is deposited are little affected, from the abscess occurring near their outer surface; and in this way we may not be able to tell before death what internal part is particularly deranged. There is no texture in the body in which this destructive secondary mischief may not ensue, and which is often the unseen, and, therefore, the unsuspected cause of death in phlebitis.

Two explanations have been offered respecting the formation of secondary abscesses—either, that they are caused by the pus being simply carried to that part and deposited there, or, that the purulent fluid, being mixed with the blood, acts as a local source of irritation on the capillaries of the part, and excites inflammation and suppuration. The reasons given in support of the former might be —that secondary abscesses do not always follow the formation of purulent matter in a vein—that pus may be received apparently into the circulation, as in the natural absorption of a common abscess, without being followed by any such disastrous effects—and

that, were secondary abscesses dependent on pus merely circulating with the blood, they ought not to be so often solitary, but appear in several parts of the body at one and the same time. On the other hand, in favour of the latter opinion it may be alleged—that the quantity of fluid found in a secondary abscess is often greater than could, by any possibility, have been separated from the blood at the part, for a large internal collection will be found where the local suppuration in the vein is trifling—that there is not simply a deposition of purulent fluid, but evident marks of violent inflammation, followed by disorganisation of the texture which happens to be affected—and that in some parts, as the eyeball, the changes which result are obviously not depending on the mere lodgment of purulent fluid, but on the destructive action caused in all probability by its presence.

Chronic phlebitis produces a deep-brown colour and thickening of the tunics; lymph is poured out, and the canal of the vessel is closed up. Sometimes ulceration of the coats takes place, and fatal hemorrage ensues, of which we have seen several examples in the superficial veins; but more frequently ulceration of veins is caused by an extension of this process from the tissue in which they are placed.

Nerves.—If we except some of the changes going on in the brain and spinal marrow, the investigation of which would be foreign to our present purpose, we may at once confess that we know nothing of the progress of inflammation in the nervous structure. We should expect that, where nerves were inflamed, their sensibility would be much increased and excruciating pain produced; but we have evidence for believing that nerves are not so often themselves the seat of disease, as that they are secondarily affected by disease in their neighbourhood. When, for example, inflammation is established in a part, we have not yet been able positively to decide, whether the pain which is experienced arises from the extremities of the nerves being directly involved in the affection, or from the pressure they sustain by the local distension, although the evidence is unquestionably in favour of the latter supposition. We can state, however, with confidence, from various phenomena observed in the living body, that a painful condition of a nerve may occur without any signs of local inflammation, and that the occurrence of pain is dependent on the healthy function of the brain and on an uninterrupted supply of nervous influence from that organ.[1] In some cases of neuralgia, where opportunities have been afforded of examining the state of the affected nerve after death, it has been stated that the neurilematous sheath was thickened, and the nerve itself enlarged; but in others, no deviation from the normal state could be perceived. In many of these we find that the real seat of disease is at the connection of the particular nerve with the brain, although pain is experienced in a distant branch; while, in all instances, the inconstancy of the affection, the often-disturbed state of the general

[1] See Local Symptoms—Pain.

health, and the nature of the remedies which afford relief, show that inflammation is not the cause of the disorder.

When nerves are partially divided, the injured portions will be occasionally found red and swollen, and the irritation from which has been supposed, in some cases, to account for the invasion of tetanus. Mr. Liston[1] has related an instance, where, during an attack of tetanus from injury to the hand and wrist, amputation was performed, and in which the median nerve was found partially divided, red and swollen; and we have seen the same thing in the communicating tibial, from a pebble shot into the calf of the leg. Yet, although these may account for such a train of symptoms in some cases, they will not in all—as evinced by the lapse of time after the injury, and the healing condition of the wound. When either of the cutaneous nerves, from taking a more superficial course, is wounded in venesection at the head of the arm, slight neuralgic symptoms will remain for long after; but no other bad effects, as far as we have seen, ever result from the injury. When nerves are completely cut across, as in amputation, their extremities sometimes expand into a substance resembling nerve, giving rise to the most painful sensations, and preventing the slightest pressure being made on the face of the stump. The newly-formed matter is firm and white, and varies both in figure and size; it is intimately connected with the extremity of the parent nerve, which is indurated and enlarged, and has been supposed to be the result of chronic inflammation. Mr. Langstaff has shown, however, that the painful condition of some stumps is owing to the extremity of a nerve getting involved in the hardened cicatrix; and to prevent which, as nerves do not retract like muscular parts but hang out at the wound, he recommends a portion of them to be cut away. The small tubercle developed in the course of a nerve, and productive of such extraordinary pain when handled, may likewise be owing to some species of chronic inflammation; but our imperfect acquaintance with the pathology of the nerves prevents us from arriving at any accurate conclusion on this and many other points wherein they are concerned.

Lymphatics.—The lymphatic vessels, with their glands, are often affected with inflammation; and, when we consider their number and the function which they have to perform, we need not wonder that such should be the case. Those placed superficially are more frequently affected than those which are deeper seated. Inflammation of the lymphatics is generally to be attributed to the absorption of some fluid, as pus, or a poison of animal origin; in susceptible constitutions, the disease will arise, also, from the slightest irritation, as the prick of a clean needle. It is rare, however, that inflammation of the absorbing vessels follows ordinary wounds; for, where it appears after blood-letting, we find that there is always suppuration around the opening made in the

[1] Elements of Surgery.

vein, and to the absorption of some of the matter from which the affection is to be ascribed. In such instances the inflammation spreads upwards and downwards, or towards the termination and commencement of the absorbing system. When the superficial lymphatics are inflamed, they form red lines running under the skin, feel like a piece of whip-cord to the finger, and may be traced as far as the glands in which they terminate. On dissection, their coats are found red and thickened, their canals filled with an adhesive matter, and the cellular tissue in which they are imbedded infiltrated with a serous fluid. The absorbents of the uterus and the receptacle for the chyle have been seen filled with a purulent fluid; and the researches of Tonnelle and Duplay in France, and of Dr. Lee in this country, have proved that inflammation of this system of vessels and of the thoracic duct occurs in puerperal women, and gives rise to as disastrous consequences as uterine phlebitis.

The glands may be the seat of acute inflammation without the absorbing vessels participating in the morbid action, of which the syphilitic bubo forms a familiar example. In such instances, the local symptoms are strongly marked, and the constitution sympathises or not according to the extent of the mischief. The affection may be limited to one gland, or extended to several. The great swelling which occasionally ensues arises from the infiltration into the surrounding cellular membrane, for the ganglions themselves are but little enlarged. The effused fluid consists of serum and lymph; and, from the large quantity of the latter sometimes deposited, the part remains indurated long after the inflammatory action has disappeared. On examination of an inflamed gland, we find it increased in size, of a spongy consistence, of a red or nearly violet hue, and exhibiting a number of similarly coloured spots when cut across. Mercurial injections may be made to pass through the convoluted absorbents of which the ganglion consists; and from which it would appear that the canals of these vessels are still pervious. Acute inflammation of a lymphatic gland may end in resolution; very frequently it runs into suppuration, and particularly after the absorption of certain substances. The purulent fluid is chiefly deposited in the inflamed cellular tissue surrounding the gland; for when a large opening is made into the seat of the disease, or the parts slough extensively, the ganglions may be seen of a dirty ash colour, and nearly of their natural size. In other cases, the pus is effused into the centre of these bodies, when the glandular structure is absorbed, leaving only the cellular envelope, which forms a bag or cyst for the matter. Where the local action is severe, occurring particularly in a bad habit of body, or one in which mercury has been unsparingly administered, great destruction of the cellular tissue, and sloughing of the integuments arise, accompanied with rapid prostration and general sinking of the vital powers.

The lymphatic glands are frequently the seat of chronic inflam-

mation, occurring either primarily or as the remains of that which had been more acute, but had not proceeded to suppuration. The parts are swollen, stiff, and hard; and, in the groin or arm-pit, the disease is a source of much inconvenience. The cellular tissue is filled with lymph, which forms a dense capsule to the inflamed gland; and the latter, on being cut into, presents a brownish structure, intersected by numerous blood-vessels. This variety is often met with as a primary symptom of syphilis. The induration may remain for a great length of time before it disappears; or a more acute attack of inflammation supervening, suppuration ensues, and the disease is thus got rid of.

An important modification of chronic inflammation in the lymphatic glands occurs in individuals of a strumous or scrofulous constitution. The frequency with which this form of disease is observed, has led some to adopt the opinion that scrofula had its seat in the absorbing system; but it is more correct to say that this system partakes only in common with other parts, and experience has now amply demonstrated that, with the exception probably of the muscular, there is no texture or organ which may not, occasionally, be the seat of this variety of morbid action. It is not our duty to enter on the general pathology of scrofula, but merely to detail its effects in certain structures; we cannot define what this state is, we can only point out its general character, and the habit of body in which it is manifested; for its hereditary descent, and other inherent features, show it to originate from a peculiarity of constitution which is received at birth, and which has the power of modifying disease arising from ordinary exciting causes. The most remarkable circumstance connected with scrofulous affections, and one which is peculiarly characteristic of their nature, is their remaining indolent for a length of time at their commencement, and afterwards assuming a more active character, during which they run into suppuration or ulceration. Healthy inflammation, however, may not only arise in scrofulous subjects, but may coexist with scrofulous disease; neither are we warranted in calling every indolent or protracted disease by the name of scrofula.

The absorbent glands most frequently affected with scrofula are, first, those about the neck and jaw; secondly, those found between the folds of the mesentery; more rarely, those at the groin; and still more rarely, those placed internally. It has been a question whether scrofulous action in a gland occurs primarily, or whether it does not proceed from disease in the neighbourhood in which its absorbing vessels arise. When the glands about the neck are affected, the exciting cause is, in general, cold, or local irritation, as from dentition; when those of the mesentery are the seat of scrofulous inflammation, the enlargement may proceed, in some instances, from disease of the mucous membrane of the intestines, or the absorption of vitiated chyle; but there are others in which its occurrence seems spontaneous, or, at least, there is no local source to which we can with propriety refer it.

Scrofulous inflammation of a gland is peculiar to childhood, and seldom met with after puberty; but we have seen disease of these bodies strongly resembling it, and which we attributed to vaccination. Within two or three weeks after the application of the vaccine lymph, the diseased action commences; glandular swellings arise in the groin, arm-pit, and neck, proceeding to suppuration; ulceration occurs over the joints of the fingers and toes, extending to the bones, and causing caries; accompanied with general emaciation and a cachectic state of the system. In one child, at the age of four years, the growth had stopped at the end of the tenth month, while the disease continued to progress. The situation and extent of the local mischief—the period of its invasion, usually within a short period after vaccination—and the absence of any marks of scrofula in the parents or other children, can leave little doubt, in an unprejudiced mind, as to the nature of the cause; nor can we hesitate to deny that the introduction of vaccine lymph may not, under certain circumstances, act as an animal poison.

Scrofulous inflammation may be confined to one gland, or spread from one to another, till it affects the whole group in the vicinity. In scrofulous inflammation, the gland itself is slowly enlarged, of a dusky brown colour, and more vascular; when cut into, a peculiar gray-coloured substance like mortar is seen deposited in different parts of the tissue, forming the proper scrofulous tubercle to the softening and future changes of which the subsequent disorganisation is to be attributed. The local symptoms are swelling; increased heat, perceptible on examination of the hand; more or less pain on pressure; and with an absence of constitutional fever. The general health, however, in most cases, is disturbed; the skin feels harsh and dry; the tongue covered with a brown fur; the breath fetid; the evacuations from the bowels unnatural; and the appetite depraved.

If the inflammatory action is not repressed, the swelling continues to augment, the body of the gland is uniformly enlarged, and the tumour acquires a doughy feel, which is gradually exchanged for one of fluctuation. The skin over the swelling often takes on a livid tint; and, if an incision is now made into the gland, a thin fluid, mixed with a peculiar curd-like substance, is discharged. If no artificial opening has been made, the skin at one part turns darker, next it is thinned and ulcerates, and then the contents of the abscess are evacuated. In general, the ulceration extends a considerable way over the gland, and the integuments are so loose and detached that a probe may be insinuated around and beneath them. After the matter is discharged, the cellular tissue which formed a capsule to the ganglion remains as a cyst, and secretes a thin whitish fluid; the skin retains its red or pink appearance, and un untractable and unseemly sore is left behind. Sometimes the abscess bursts in several places, and bridles of diseased skin cross the surface of the ulcer in different

directions, from beneath which the discharge oozes. An ulcer thus formed shows no disposition to granulate, but remains stationary, and secretes a thin glairy fluid; the skin around retains its red colour; and, should cicatrisation take place, the integuments are so much puckered and drawn together that considerable deformity results. In some instances, the glandular swelling continues indolent; the skin does not change its colour; no uneasiness is experienced from handling the parts; and, after a time, the enlargement subsides. On the other hand, whenever the tumour turns painful, begins to soften, and the skin over it puts on a dusky red appearance, we may be assured that imperfect suppuration will follow. Scrofulous inflammation never terminates in mortification, but often in ulceration. It is, in fact, a chronic disease, modified by a peculiar habit of body, and is often remarkable for its indolence at one time and its sudden activity at another. The changes arising in the lymphatic glands from scrofula, especially in those about the neck in females, are of great importance to the surgeon, both from the frequency of their occurrence, and the deformity resulting from unskilful treatment.

Serous Membrane.—Under this head we include the arachnoid of the brain, the pleura and pericardium in the chest, the peritoneum in the abdomen, and the tunica vaginalis of the testicle. These membranes, in the natural state, are thin, shining, semi-transparent, lubricated by a fine fluid on their inner surface, and connected to the subjacent parts by cellular tissue; when affected with inflammation, they lose their brilliancy and transparency, become opaque and thickened, and numerous vessels, carrying the coloured portion of the blood, ramify throughout their structure. In studying the effects of inflammation in this class of membranes, it is necessary to pay particular attention to the cellular tissue by which they are connected to the subjacent parts. It is in this connecting tissue the first indications of inflammation are to be found, and to which the morbid appearances are confined, should the disease prove fatal within a few hours after its invasion. It varies in quantity in different situations, and even in different parts of the same cavity; in some, loose and abundant, as between the arachnoid and pia mater; in others, more dense and sparing, as between the former and the dura mater. This variety in the distribution of the cellular tissue modifies the effects of inflammation in serous membranes, and is a point to which the attention of the pathologist should be strongly directed.

The commencement of acute inflammation in a serous membrane is denoted by the infiltration and injection of its subcellular tissue with red vessels. As the morbid action goes on, the infiltration increases; the serous fluid penetrates between the layers of the serous membrane itself, and causes increased thickness, with loss of transparency. The opacity, at least at first, is not owing, as has been generally supposed, to the natural exhalation from the surface of the membrane having ceased, but to the fluid effused between its

layers and into the connecting cellular tissue. These changes are best seen by dissecting off the membrane, and placing it between the eye and the light. As the morbid action spreads from the adherent to the free surface of the membrane, red vessels appear in solitary parts, forming lines which coalesce, and leaving the intervening portions of the tissue opaque and thickened. When the inflammation is very intense, some of the vessels occasionally burst, by which blood is poured out, and which has been erroneously imagined to be a secretion. There are some serous membranes, as the arachnoid, which seldom admit of being injected with red blood, and others in which the redness is not permanent; so that the most obvious signs of inflammation, in this class of structures where the disease has terminated early, are their milk-white appearance, loss of transparency, increased thickness, and infiltration of a serous fluid into the subcellular tissue.

Inflammation may thus begin and end without any further changes; but, in general, as soon as the disease reaches the free surface of the membrane, the natural exhalation is suspended, and new products of different kinds are formed. These may consist of a serous fluid of different colours of lymph, or of purulent matter; and one or all may occur together in the same case. When the inflammation is moderate, the effused fluid is either clear and limpid, or slightly lemon-coloured; it is made up of water, holding a small quantity of albumen in solution, and from which its slightly alkaline property is probably derived. When the morbid action is more acute, the surface of the membrane is coated over with a soft plastic substance, susceptible of spontaneous coagulation, and found, on analysis, to be fibrin mixed with a small quantity of an albuminous fluid. As the thinner part is absorbed, the effused substance becomes more adhesive, but may readily be scraped off, so as to leave the surface of the membrane smooth beneath. This lymph or fibrin—known by the name of adventitous, false, or pseudo-membrane—is afterwards penetrated by blood-vessels, and unites the opposite surfaces of the tissue on which it is effused.

The period within which the false membrane is organised seems to vary extremely. In some cases, vessels have been discovered in less than twelve hours from the supposed effusion; in others, no vascularity could be detected after a lapse of several months. It is not improbable, that much of this discrepancy depends on the unsuccessful manner in which the examination has been made; for the vessels in the clot of blood have been successfully injected both by Mr. Hunter and Sir Everard Home. The vascularity is first indicated by a number of red dots, resembling the "*punctum saliens*" in the vitellary membrane of the chick, which run together and form lines, communicating frequently, and traversing the membrane in various directions. These red lines are soon observed to be the rudiments of the future vessels through which the blood regularly circulates, and which increase in number as the organisation becomes more complete. It is still undetermined, however, in

what manner the newly-formed membrane acquires its vascularity; whether the vessels shoot into the effused lymph from those of the serous membrane beneath; or, whether they are not first formed in the lymph by an independent vitality inherent in itself. The former is the opinion generally entertained, and is supported by the recent microscopical researches of Gendrin; the latter has been insisted on by Laennec, who considers that portions of effused fibrin perform the functions of secretion, absorption, and nutrition, before any trace of vessels can be discovered. In whichever of these two ways the vascularity is first effected, we observe that, after its completion, nerves and absorbents supply the new formation; next succeed those changes by which the false membrane resembles the tissue on which it had been thrown out, becomes itself susceptible of taking on the process of inflammation, and gives origin in turn to similar exudations.

Where lymph is effused on the surface of a serous membrane by inflammation, we frequently find that other fluids are poured out along with it; but, in some rare instances, the exudation of fibrin takes place alone. Very frequently masses or shreds of the lymph are detached, and seen floating in the other fluids which are present. In such cases, the serum is generally turbid, or has a slight sanguineous admixture, yields on examination more albumen, and shows stronger alkaline properties. When the inflammatory action is greater, and has continued for several days, the serum becomes highly alkaline, acquires a greenish or yellowish colour, and often contains a considerable quantity of pus, which falls to the bottom, and gives out an offensive smell. In the most acute form of the disease, mortification ensues, and, in some cases, with great rapidity. Primary ulceration of a serous tissue never occurs as an effect of acute inflammation.

Serous membranes are very liable to be affected by chronic inflammation, and in them the disease gives rise to different results. It is during its long continuance that we find the opposite surfaces of these tissues so firmly connected; as the arachnoid lining the dura mater to that covering the upper surface of the brain, the pleura pulmonalis to the pleura costalis, and the peritoneum to the intestines. From the lymph which is thrown out becoming organised, and in its turn giving origin to fresh exudations, the original membrane always appears much thickened; the subserous tissue is filled with coagulated albumen, and the vessels have a dull red or brownish appearance. When the adventitious and subcellular tissues are macerated and then carefully dissected off, the proper membrane will be found opaque, and of its natural thickness. The most extensive adhesions are in this way formed, especially in the chest and abdomen; and in the former often without any complaint of pain, or the individual being aware of the existence of the disease. False membranes, thus originating, are frequently extremely firm, and yield to the knife with as much difficulty as a piece of cartilage.

In chronic inflammation of a serous structure, adhesions may exist with or without the presence of other fluids. In some cases, particularly when the peritoneum is affected, tubercles form on the inner surface of the membrane, and which may suppurate and burst into the general cavity. Chronic inflammation may cause increased secretion from the exhalent surface of a serous membrane, and give rise to a form of dropsy. All the three great cavities are liable to this variety of disease. Purulent matter may also be poured out by a serous tissue, under long-continued passive inflammation. This is more especially met with in the chest, where it has received the name of *empyema;* and where the fluid collects in such quantities as to fill the cavities of the pleura, without any ulceration or breach of surface. In these cases we have generally found the membrane thickened and opaque, and its interior rough, and having a granulated appearance. The amount of either serous or purulent fluids, effused during chronic inflammation of a serous texture, is so various that no general estimate can even be given; in the brain it cannot exceed a few ounces, while in the chest or abdomen it may weigh several pounds.

The local and constitutional symptoms are probably better marked during acute inflammation of a serous membrane than in any other texture of the body, and usually correspond to the intensity of the morbid action and the strength of the patient's constitution. The effect on the system, however, varies with the particular seat of disease. Thus, in the head or chest, the pulse is *full and bounding*, and the symptomatic fever of the sthenic type; while in the abdomen the former is *small and wiry*, and there is apparently great depression of the vital powers. This discrepancy can only be ascribed to the nerves with which the peritoneal membrane is so freely supplied, and which are derived from the ganglions of the great sympathetic; in all such cases, too, the pulse becomes rounder and softer after free depletion.

Mucous Membrane.—This tissue lines all the different outlets of the body, and is extremely liable to become inflamed. When we consider the extent of surface over which this membrane is diffused, and the numerous sources of irritation to which it is exposed, from external as well as internal impressions, we need no longer wonder at its being so frequently the seat of disease. Thus we have a mucous tissue lining the eyelids, nose, mouth and throat; the trachea, to its minutest subdivisions; the œsophagus, stomach, and the whole of the intestinal canal; the ducts and pelvis of the kidney; the ureter, bladder and urethra; and in the female, the uterus and vagina. The mucous membrane is far more extensive than the external covering of the body to which, in so many respects, it is allied; nay, it has even been calculated that the lining of the bronchiæ, if unfolded, would exceed by several times, the square surface of the skin; and hence the free channel opened up for the absorption of the various atmospheric poisons.

In its natural state, the mucous membrane is pale; but imme-

diately on inflammation taking place, it becomes injected with red vessels. Its structure is so much more lax than that of a serous membrane, that red blood at once circulates through it; hence redness is the earliest sign of incited vascular action. In *post mortem* examinations, particularly of the abdomen, we must be careful not to mistake redness of the mucous membrane from other causes, for that arising from inflammation; the former is evanescent; the latter is unaffected by pressure or position, and does not disappear after maceration or drying. The brightness of the colour varies with the acuteness of the inflammation; in the most vehement species, the membrane is of a scarlet tinge. Immediately after the morbid action has commenced, the submucous cellular tissue is injected and infiltrated with a reddish serum, which, along with the increased vascularity of the membrane itself, gives rise to swelling. In some cases, the tumefaction thus created is the cause of very formidable symptoms—as in ophthalmia, to protrusion of the conjunctiva and elongation or the eyelids; or in laryngitis, to narrowing of the glottis and impending suffocation.

Derangement of function is also one of the first indications of inflammation in the mucous membrane. Its natural secretion in health consists of animal mucus, held in solution by a watery fluid; unlike that from a serous tissue, it is readily discharged from the system; hence its examination becomes valuable where the actual disease of the membrane is concealed from view. The first effect of inflammation is to increase the secretion and convert it into a thin, transparent, or brownish-coloured fluid, possessing, with the exception of that from the alimentary canal which always remains acid, slight alkaline properties. These changes are best exhibited by dipping a piece of turmeric or litmus paper in the discharged fluid. Frequently, the secretion is rendered more acrid from an increase in the quantity of soda, excoriating the parts over which it flows, and also coagulating, when thrown into boiling water, from the albumen it contains. Sometimes, at this stage, the secretion is tinged with blood, or some of the over-distended vessels burst, and pure blood is discharged; and if the latter is in any quantity, it usually has the effect of relieving the local complaint. As the inflammation increases, the secretion diminishes, and is sometimes completely suspended when the disease is at its climax; in other cases, the discharge, though diminished in quantity, continues during the whole period. As the inflammation begins to decline, the secretion returns, altered, however, in character; being a mixture of transparent mucus and yellow pus, or, as it has been named, puriform-mucus. The proportions of pus and mucus may be pretty accurately ascertained by the viscidity of the discharge, and by the common test of throwing part of it into water. If mucus abounds, the fluid is tenacious, ropy, and floats on the surface of the water; if pus predominates, the secretion is less viscid, and it sinks to the bottom of the vessel. When the inflammation has been very acute, the secretion is principally puri-

form; and, as the disease declines, the mucus again becomes more copious. In all cases, the increased discharge has the effect of relieving the congested state of the membrane.

Where puriform matter is furnished by a mucous membrane, no ulceration, under ordinary circumstances, is present; there is a mere alteration in the mode of secretion, as is proved by the return to mucus as the disease declines, and by no breach of surface being descernible on examination. It may be stated, as a general law, that mucous tissue shows a greater disposition to run into suppurative inflammation; serous, on the contrary, is more prone to take on the adhesive mode of action. This is a provident arrangement in nature; for, had the phenomena been reversed, we should have had, in the one case, outlets obstructed; and, in the other, secondary effects proving almost invariably fatal. When, by the violence of the local action, false membrane is thrown out on a mucous surface, we generally find that a fluid is effused between them, by which their adhesion is prevented and the effused lymph may be discharged. We have examples of this in croup, where the false membrane is coughed up; and occasionally, in virulent gonorhœa, where shreds of lymph, resembling the urethra in shape, come away with the urine. In the experiments recorded by Mr. Hunter, it required the most violent means to induce the adhesive inflammation in a mucous structure. In some instances, the mucous membrane is found in a state of mortification; it is then easily lacerated and detached, and gives out a fetid smell—from both which signs we are enabled to distinguish it from congestion or extravasation of blood into the subcellular tissue.

The general effect of acute inflammation, in a mucous membrane, is to render it soft, pulpy, and easily scraped off from the adjoining tissue. The disease usually commences in distinct patches, occupying only a small extent of surface and leaving the intervening portion free; sometimes the inflamed spots run together, and the affection spreads rapidly along the membrane. In what has been described by Bretonneau, under the name of *diptherite*, the inflammation first appears in the fauces, but quickly extends upwards into the nasal fossæ, and downwards into the trachea and bronchiæ, and proves fatal within a few hours after its commencement. Suppuration frequently occurs in the mucous follicles when the membrane lining the mouth and throat has been inflamed, as in certain forms of fever; and these abscesses have been long known under the title of aphthæ. Pustules are said likewise to be found in the intestinal canal, when the skin is the seat of small-pox.

Chronic inflammation of a mucous membrane occurs in solitary spots of a brownish colour, leaving the intermediate portions of the tissue free and of the natural appearance. In this form of disease, the membrane is sometimes softened, pale and thin, as we have found in bronchitis; more generally, it is indurated and thickened, and its vascularity is distinctly increased. In cases of long standing, the induration and thickening arise from effusion of an albu-

minous fluid among the interstices of the membrane and into the subjacent tissue; the parts are firm to the touch, and cut with the knife like cartilage, as in old permanent stricture of the urethra. The secretion from the membrane consists of a viscid, ropy mucus, with or without admixture of purulent matter—the quantity of the latter varying generally with the degree of inflammation present. A small extent of membrane will sometimes furnish a copious mucous discharge. In catarrh of the bladder, the vessel containing the urine will be half filled with mucus, so tenacious as not to fall out when the vessel is inverted. In such cases, the system speedily sympathises with the local disorder, and the general health breaks up.

Ulceration is a frequent effect both of acute and chronic inflammation in a mucous membrane, and occurs with greater facility in some situations than in others. Its most frequent seat is in the intestines, and next in the urinary organs; while, with the exception of the nostrils, throat and larynx, the rest of the respiratory apparatus may be considered exempt. The ulcers assume different characters, and vary in depth, extent and number. When the ulcerative action is acute, a considerable portion of the membranous surface may appear abraded; or the ulcers may be small, distinct, and with irregular edges. The ulceration may be confined to the mucous membrane or extend to the neighbouring tissue; in the intestinal canal, the muscular and peritoneal coverings are thus penetrated, and the fæcal matter escapes into the general cavity of the peritoneum. The perforating ulcer in the intestinal canal is more frequently the result of acute than chronic inflammation, and will generally be found to occupy the last portion of the ileum and the beginning of the colon. In chronic inflammation, the ulceration is more sluggish; the edges of the sores are better defined, being tumid or inverted from the thickening of the subcellular tissue. Although ulceration of a mucous membrane is in many cases a formidable occurrence, we have not, with few exceptions, any distinct proof at what period it may have taken place. The appearance of pus in the discharge is no indication of such a change, because it is a peculiar property of these tissues to have their secretion for a time altered, and converted into one of a purulent character. In inflammation of the mucous lining of the intestines, where there is a sudden increase of pain with great distension of the abdomen, a feeble pulse, and rapidly-approaching dissolution, we have reason to fear that an ulcer has penetrated the coats of the viscera, and allowed some portion of fæces to escape. In catarrh of the bladder, again when ulceration has occurred, the sufferings are much augmented from the urine coming in contact with the abraded surface.

When mucous membranes are inflamed, their natural sensibility is only slightly increased; the pain, even in the most acute form, is trifling compared with that arising from a serous tissue, and is generally of a dull and prickling character. We endeavoured to

explain this difference when treating of pain as a local symptom. The increased sensibility of mucous textures appears likewise to be principally called forth, when any of the fluids which wash over them in their natural state come in contact with them when inflamed. This symptom, independent of all others, is valuable to the medical practitioner, as showing him that some portion of the membrane is in a morbidly irritable condition. When the mucous coat in the lower part of the intestines is inflamed, pain, tenesmus, and griping, are present before an evacuation, after which a corresponding feeling of relief is experienced. Should the bladder be the seat of disease, there is frequent desire to empty it, and great uneasiness while the urine is collecting; in gonorrhœa, the pain, from the urine passing over the inflamed surface, is often extreme. The fever accompanying inflammation of a mucous membrane partakes more or less of the typhoid character; nor in any form we meet with, do we find the well-marked sthenic variety which occurs during a similar state of a serous tissue.

Fibrous Tissues.—Under this general head, the anatomist includes the fasciæ about the joints, those binding down the muscles in particular situations, as in the fore-arm or thigh, tendons, ligaments, periosteum and aponeuroses of all kinds; but whatever may be their natural structure, pathology teaches us that many of them are at least distinct as they relate to disease. Inflammation, either acute or chronic, of the fibrous tissue around the joints, or of the aponeurotic expansions covering certain muscles, as those of the back, constitutes true rheumatism—an affection peculiar both in its nature and treatment. However violent or long continued the local action may be, we rarely or never find that it proceeds to suppuration; the most ordinary effect is an effusion of lymph, with thickening and rigidity of the tissue, and by which the motions of a joint may be much impaired. The disease seldom attacks the muscular fibre; and when muscles are in this way made to participate, their affection is entirely secondary. Rheumatic inflammation is remarkable for its tendency to recur in some form or other; and, in certain constitutions, its attacks are apparently much influenced by the temperature of the weather. It often assumes an erratic form, leaving its original seat, and wandering from joint to joint, till it travels over the different articulations of the body. In some cases, the inflammation translates itself to an internal part of similar structure, as the membranes of the brain, but more generally the pericardium or pleura; and it is not uncommon to find both the external and internal affection existing at the same time. Such cases have been described as a metastasis, or translation of the disease; and in connection with chronic rheumatism, we have seen the surface of the heart studded with small nodules of lymph, firmly imbedded in the substance of the membrane, and giving to it a rough and granulated appearance.

Rheumatic inflammation of a joint is marked by redness and slight swelling; the pain is aggravated during the night or on

motion, and in some cases by preternatural heat, and in others by exposure to cold. During the symptomatic fever consequent on an acute attack, the functions of the brain never suffer; there is usually a moist state of the skin and tongue from the commencement; the blood is buffed out of all proportion to the exigency of the case, and remains so throughout. Inflammation, again, of such fibrous tissues as those of the forearm or thigh, and which may ensue spontaneously or after wounds, manifests no disposition to change its seat; the local as well as the constitutional symptoms run high; the swelling and tension are very great, and suppuration generally follows. In the worst cases, mortification succeeds; the membrane changes to an ash colour, loses its vitality, and sloughs away in shreds. Ulceration never occurs as a primary effect of inflammation in fibrous textures.

Muscular Fibre.—Muscles are rarely the primary seat of inflammation; and, considering their great abundance in the body, it is a fortunate provision. They are sometimes secondarily affected by the disease spreading to them from their coverings, as we observe in rheumatism and enteritis. One of the most curious effects from inflammation is a motionless state of the muscle; the fibres are paralysed, and no irritation of the nerve supplying them, or even the application of galvanism, will cause their contraction. We have an example of this in enteritis, when the inflammation spreads from the serous to the muscular covering of the intestines; obstinate constipation is then present, and all attempts to relieve it by purgatives are both fruitless and improper.

When muscles are injured by external violence, by the sharp fragment of a fractured bone, or, in the lower animals, by a powerful stimulus, we have then an opportunity of watching the effects resulting from inflammation. There is first an increased influx of blood, by which the muscular fibres are rendered of a deeper colour, and the cellular tissue is injected; next follows an effusion of serum and lymph, agglutinating the substance of the muscle and increasing its bulk. Some of the over-distended vessels burst, blood is effused amongst the cellular interstices, and the proper muscular fibre is softened. If the inflammatory action is more violent, in addition to other fluids, pus is deposited in different parts of the muscular structure, which now loses its cohesion so completely as to lacerate in all directions on the slightest touch. Should mortification ensue, the fibres turn black or green, readily tear, and slough away in detached shreds. When a portion of muscle is thus lost or purposely cut away, its place is supplied by an adhesive matter of great strength, which serves as a bond of union between the separate ends, but which never assumes the appearance or action of the original fibre. Ulceration never occurs as a primary effect of inflammation in muscular fibre; and, when muscles are engaged in this process, as we observe in the heart, extremities, &c., it is from the ulcerative action spreading from an adjoining tissue, and involving them in its progress. The

tendons of muscles, though exhibiting neither sensibility or vascularity in the normal state, are, nevertheless, susceptible of becoming inflamed. Under this process they are slightly reddened, lose their glistening appearance, and assume a dull or dead white; their cohesion is so far destroyed as to make them plastic to the touch; their vitality is readily extinguished, and they always separate at their connection with the muscular fibre.

Synovial Membrane.—The synovial, like the serous membranes, form shut sacs, have a shining or polished appearance on their inner surface, and secrete a glairy albuminous fluid which lubricates the joints. They are subject to inflammation of an acute or chronic form; and those which line the knee or ankle, either from extent or having the thinnest covering, are the most frequently attacked. Acute inflammation usually commences in one spot, but quickly spreads over the rest of the membrane; the cellular tissue on the outside of the joint is infiltrated with serum; the capsule itself becomes highly vascular; an increased quantity of synovial fluid, of a less albuminous quality than occurs in health, is poured out; and from all which there arises considerable local swelling. If the inflammation is violent or long-continued, coagulable lymph may be effused into the cavity of the joint; the synovial membrane may, from the same cause, be thickened, and its opposite surfaces made to adhere; but these effects are not so common as in the serous tissues. In some cases, under great local excitement, purulent matter is secreted, forming an abscess within the capsule, and making its way out by one or more ulcerated openings. Sir B. Brodie[1] considers that suppuration rarely takes place from a synovial membrane, independent of ulceration; but, although this holds true as respects chronic diseases, pus is frequently effused during acute inflammation, without any breach of surface. The constitutional symptoms, from active inflammation of a large joint, as the knee, are often exceedingly severe, the fever being principally of the sthenic type; when purulent matter forms, delirium and coma ensue, and, should the patient survive, the inflammatory is succeeded by fever of a hectic character.

Chronic inflammation of a synovial membrane is of common occurrence, and has long been described under the absurd title of rheumatic white swelling. The disease is at first confined to one part of the membrane, and slowly spreads throughout the tissue; there is an increased collection of synovia in the joint, but which is afterwards absorbed if the inflammation subsides. In cases of long standing, the synovial membrane becomes thickened, its inner surface deeply coated with coagulating lymph, and the shape of the joint considerably altered; eventually, suppuration takes place, the capsule ulcerates in one or more places, and the articular cartilages are absorbed. Where ulceration of the cartilages, however, is observed in combination with inflammation of the synovial mem-

[1] Treatise on Diseases of the Joints.

brane, it more frequently happens that the former has been the primary or leading affection—the latter only supervening on the formation of an abscess within the joint. Inflammation of the synovial membrane is not so common in young persons as in adults, a circumstance which assists us in distinguishing its more chronic forms from some other diseases incidental to joints.

Lastly, the synovial membrane is subject to a peculiar change of structure, by which its natural organisation is completely effaced. The capsule assumes a reddish brown colour, and is converted into a pulpy substance, varying from a fourth to half an inch in thickness. The disease generally involves the entire membrane, and remains indolent for a length of time; the cellular tissue on the outside is infiltrated, and there is much stiffness and immobility of the joint. As the disease progresses, abscesses form in various situations; the articular cartilages disappear; the ends of the bones become carious; and, unless the joint is removed, the patient wastes under the accompanying hectic fever. This change in the structure of the synovial membrane has been supposed by some to be independent of inflammation, but it is more probably the result of this species of morbid action in a scrofulous constitution. The knee is almost the invariable seat of the affection; it takes place in early life, and cannot be arrested by any means we are yet possessed of.

Cartilage.—This structure is neither very liable to become inflamed, or capable of long withstanding the irritation from disease. When inflammation spreads from a synovial membrane to the cartilage beneath, the latter first becomes vascular, then swollen and of a spongy consistence, and, lastly, ulcerates at one or more points. Ulceration of the cartilage of a joint may either follow from disease in its neighbourhood, or it may arise as a primary affection, and while the other parts making up the articulation are sound. Primary ulceration of an articular cartilage seems, in the majority of cases, to be independent of inflammation; but is generally preceded by a soft and spongy state of its structure. Mr. Key of Guy's Hospital has endeavoured to show that the absorption of articular cartilage is caused by the synovial membrane, some portion of which, if the joint be carefully opened, will be found of a fringed appearance, and lying in contact with the abraded surface; but, besides the proofs that cartilages possess absorbents as well as blood-vessels, we find them ulcerated in situations where no synovial membrane is opposed to them, as where the absorption commences on the side next the bone.

Ulceration, or rather ulcerative absorption of an articulating cartilage will go on for a length of time without any purulent matter being effused into the joint, and even when the ends of the bones have been rendered carious. The formation of pus, in such cases, seems to be principally dependent on the inflammation of the synovial membrane. The disease is attended with pain from the commencement, especially on the ends of the articulating bones

being rubbed against each other, while the joint retains its size and shape. As the ulceration extends, the heads of the bones and the synovial membrane become affected; abscesses form within and without the joint and open in various places, and secondary dislocation may ensue. While the ulceration is confined principally to the cartilages, and matter is not formed within the joint, there is every chance of the ends of the bones throwing out granulations, and the limb becoming anchylosed; but, where the disease has spread to the synovial membrane, and suppuration followed, the hopes of a natural cure are nearly precluded. Ulceration of the cartilages forms one of the varieties of white swelling; and, though more frequently met with at the knee, is not confined to any articulation. It is a disease peculiar to early life, and seems immediately connected with a scrofulous habit; there is no attempt ever made to regain the lost structure, and hectic fever attends throughout.

Periosteum and Bone.—The pathology of the osseous system is not well understood, and we are ignorant of the manner in which many of the changes incidental to its structurue are accomplished. Bones do not, as was once supposed, consist of laminæ or plates united together by narrow processes, but are simply made up of a reticular tissue, into the cells of which the proper ossific matter is deposited. They are furnished with vessels, nerves, and absorbents, by which they are organised and have bestowed on them the properties of other textures; they are, therefore, like the soft parts, capable of taking on diseased action and repairing injuries inflicted on their substance. The diseases of bones are only remarkable for their duration and the obstinacy with which they yield to remedial means, peculiarities which are easily explained by a reference to their structure.

In studying the diseases of bones, there are some points in their natural history deserving of being carefully remembered; one of the principal of which is, the difference of arrangement observed amongst the osseous particles. Thus, some bones, as those composing the extremities, have a dense solid wall of osseous matter enclosing a canal within, which is filled with a spongy or reticular texture, and in which the membrane ramifies that secretes the marrow. In these long cylindrical bones, the compact structure is greatest in the centre; while, towards their extremities, which are expanded to afford a larger surface for articulation, the density of the external wall gradually decreases, and the interior is filled with large cells, or, as it is frequently named, a cancellated tissue. In another class, again, as the vertebræ, or the small bones of the ankle and wrist, the spongy structure is accumulated, there is merely a thin solid plate of bone externally, and in these respects, they are made to resemble the ends of the long bones. In the broad flat bones, as those of the head or pelvis, the compact substance is of moderate thickness, forming two layers or walls, between which the spongy texture is enclosed. These varieties in the arrangement of the osseous particles have long been pointed out by the anatomist,

and they are equally worthy of attention from the pathologist, in as far as they are frequently connected with different kinds of diseased action.

The bones are closely invested, except on their articular surface, with a strong dense membrane, named the periosteum, the functions of which, in some of their diseases, have given rise to much discussion. It varies in thickness according to the age of the individual and the vascularity of the bone which it covers; its outer surface is rough, and united to the common cellular tissue; its inner surface is smooth, and connected to the bone by prolongations which line the canals transmitting the nutritous vessels. The arteries of bone are of two orders—those which are derived from the larger vessels in the neighbourhood, and those which come from the periosteum. The former enter by the large foramina seen in bones, run into the interior, and are distributed to the membrane which furnishes the marrow; the latter, which are small and numerous, pass inwards through the minute pores visible to the naked eye on the surface of the compact tissue, and are considered as the proper nutritious arteries of the osseous structure. Between the two orders of vessels, and in the dense substance of the bone, an intimate communication is kept up—an anatomical fact which, if properly considered, enables us to explain the re-adhesion of the periosteum in cases where it has been stripped off and again laid down; and why the surface of the bone may not perish but throw out granulations, where its investing membrane has been detached. The vascularity of bones, as well known, is greater in youth than in adult age, a circumstance which likewise explains several points in their pathology. The presence of lymphatics in the osseous structure is proved by the absorption going forward in certain diseases, and by the common experiment of feeding an animal on madder; while the existence of nerves, besides being capable of demonstration, is plainly indicated by the pain experienced during an attack of inflammation. In studying the diseases of bones, therefore, we must particularly keep in recollection the difference in the distribution of the osseous particles; their being invested with a fibrous membrane without, and lined by a medullary one within; and the nature and mode of their vascular supply.

Bones, with their investing membrane, may be the seat of either acute or chronic inflammation. The former frequently arises spontaneously or from external injury; the latter is often connected with a cachectic state of the system, induced by scrofula, the previous existence of the venereal disease, or the unsparing use of mercury. From the unyielding nature of the structure, and the great resistance offered to the distension of the vessels or effusion of any kind, the most excruciating pain is experienced in the acute form of the disease; while, in the chronic variety, and especially that connected with syphilis, the pain is observed to remit during the day and increase towards night. Acute inflammation of the osseous structure is always accompanied by severe constitutional disturbance, and by

considerable hardness and tumefaction over the seat of disease. It is most frequently met with in the superficial bones, as those of the leg or cranium; but those which are well protected by soft parts, as the thigh, are nearly equally liable to be affected. In some cases, acute inflammation has attacked at once almost all the bones of the body, and proved rapidly fatal from the febrile disturbance which ensued.

The periosteum is more liable to be inflamed than the bone itself, and may exist as a distinct disease without the bone participating. In such a case, the inflammation is usually more active and more rapidly developed. From an intimate connection, however, subsisting by blood-vessels between the periosteum and the outer surface of the bone, the inflammation soon spreads from the one to the other, although either for a time may remain distinct. When the parts are examined, the blood-vessels of the affected bone appear numerous and enlarged from their distension with red blood, the osseous structure is looser and more cancellated, and the foramina, for transmitting the small arteries, larger and more distinct. The bone lies imbedded in an effusion of lymph and serum, poured out from the inner surface of the periosteum, which membrane is also thickened and more vascular. The inflammatory action is frequently communicated to the cellular tissue external to the periosteum, lymph and other fluids are copiously deposited, and the limb, from this cause, is much swollen and indurated. When a superficial bone, as the tibia, is thus affected, the integuments generally share in the morbid action, they become red and glistening, and the case is apt to be mistaken, by a careless observer, for one of phlegmonous erysipelas. When acute inflammation takes place in the interior or medullary texture of a bone, the pain is deeper seated, there is much less external swelling, the medullary membrane is thickened, and the bone itself enlarged. From the free vascular communication between the vessels of the compact tissue and those supplying the medullary cavity, the disease usually extends through the osseous substance and then involves the periosteum.

Acute inflammation may be limited to a small portion of a bone, with its periosteal covering, or it may extend from one articulating extremity to another. The disease terminates either in resolution, effusion of lymph, formation of matter, or death of the osseous substance. When a bone is the seat of violent inflammation, it rarely happens that a complete termination by resolution ensues; the morbid action spreads rapidly to the periosteum, and more or less lymph and serum are poured out. The lymph effused beneath the periosteum remains and becomes organised, ossification next commences at different points and gradually extends throughout the newly-deposited substance, and by which the figure of the original bone is destroyed. While the process of ossification is going forward, the outer surface of the old bone is removed, but the absorption never takes place to such an extent as to restore the former appearance of the limb. Such cases have been sometimes described

under *exostosis*, but improperly, for they are always the result of inflammatory action, and do not, in the least, resemble that slow growth of bone upon bone to which the term exostosis should be strictly limited. This termination of acute inflammation is, therefore, readily known by the alteration in the size and figure of the bone.

Suppuration frequently follows incited vascular action of a bone, either where the disease is external, and has involved the periosteum, or where it originates in the medullary or cancellated tissue. The formation of matter in all these situations is attended with severe constitutional sufferings, proceeding, of course, from the unyielding nature of the textures in which the pus is deposited. When collected between the periosteum and bone, fluctuation can be so distinctly felt that no error need be committed; yet we have known the effusion of lymph mistaken for it, and useless and painful incisions made into the swelling. When secreted into the medullary cavity, there is no local symptom to warn us of its occurrence; the matter is too strongly protected to be felt externally, and often we are not aware of its presence till on amputation or examination of the bone after death. When suppuration again occurs in the cancellated tissue, there is deep-seated pain; the bone becomes enlarged and the periosteum thickened; the reticular cells are broken down from the purulent fluid undulating through them; the external compact shell is absorbed; and then the absces breaks, leaving a deep chasm from which the discharge oozes out. This variety is common in the ends of the long bones, the lower jaw, and the cranium; it has received the name of *spinaventosa*, and been frequently, though erroneously, reckoned of a malignant character. The compact shell of bone is with difficulty removed by nature, and the purulent collection often gravitates in all directions. We have an example of this after injuries to the head, where pus is deposited in the diploe, and the abscess has not been opened by perforating the outer table of the cranium. In such a case, the fluid may pervade nearly the whole of the concellated structure of the bones, bursting by numerous small openings beneath the pericranium on the outside, and over the dura mater on the inside; and the skull, when macerated and dried, presenting the appearance of a sieve.

Suppuration, either on the external surface of a bone in its medullary cavity or its cancellated tissue, is, as might be expected, more common than in its compact substance, which does not admit of such a change till opened out by the previous inflammation, and which is at all times more liable to lose its vitality. When acute inflammation, followed by suppuration and ulcerative absorption, takes place in bone, more especially where the disease has been the result of violence and is confined to the external surface, we generally observe that the constitution and the local parts together possess sufficient powers to repair the breach which has been made; granulations spring up from the osseous structure, and unite with those from the surrounding parts in filling up the chasm. In other

cases, particularly where the cancellated ends of long bones are affected, the disease, though originating in the way we have mentioned, gets into an indolent state, secretes an unhealthy sanious fluid, and reparation is obtained with the utmost difficulty, either by nature or art. During the suppurative inflammation of a bone, the symptomatic fever always runs high, and is similar to what attends the formation of matter in the soft parts; while, after this stage is over, and the disease acquires a lingering character, hectic supervenes, under which and the local discharge the general health may completely break up.

Violent inflammation of the osseous structure, whether originating spontaneously or from external injury, is frequently followed by a loss of vitality. The word *necrosis*, from νεκρόω, to destroy, is employed to denote the death of a bone, and is used in the same sense as the term mortification applied to the soft parts. It has been justly doubted whether so vehement inflammatory action as that which occasions the death of laxer tissues ever takes place in bones. Although organised like other parts of the body, yet organisation is of a low order; the natural powers inherent in bone are weak, and any excess of action which causes obstruction to the circulation through the minute vessels is likely to end in the death of the texture itself. Hence the reason why the least vascular portion of the bone, or its compact tissue, is the most disposed to perish, and why necrosis is more frequent in the shafts than in the extremities of the long bones—in the flat than in the round ones. As a general law, therefore, it might be stated that, from acute inflammation, the dense osseous structure is more subject to necrosis, and the cancellated or spongy portion to suppuration. During the different stages of necrosis, or the separation of the old bone and the formation of the new, there arise some of the most curious and interesting phenomena which can occur within the animal economy; but we will return to this subject at a future period.

While suppuration and necrosis are to be traced to acute inflammation in a bone, interstitial deposition and ulceration are the common results of a more chronic form of disease. It is during the continuance of this latter state that we find bones becoming gradually enlarged, there being an increased deposition, not merely on their surface, but, in many cases, throughout every part of their parietes. The inflammation and swelling may be confined to a small space, or affect a large portion of the bone, producing an unyielding and incompressible tumour accompanied by a steady and sometimes gnawing pain, and leaving, after the inflammation has ceased, more or less permanent disfiguration. When the disease is limited, it frequently appears as a secondary symptom of syphilis, and is then named a *node*. In such cases, the bones which lie nearest the surface are the most liable to suffer, as those of the shins, elbows, and head; and, not unfrequently, different swellings exist on these at the same time. We have generally observed that the venereal node commences in the periosteum; this membrane becomes in-

flamed and thickened; the disease then spreads to the outer surface of the compact tissue, and lymph is effused, which is ultimately organised and converted into bone. Venereal nodes, however, are of two kinds—the one occasioned by acute inflammation, which may end in suppuration; the other, the result of slow chronic action, followed by effusion of lymph and subsequent formation of new bone. In either case, they occur on the superficies of the dense bones which are least protected by soft parts; and seem, as we have said, to originate in the periosteum.

Chronic inflammation, with enlargement, may occur in any of the bones of the body, new ossific matter being deposited throughout every part of their substance, and by which the medullary canals and cancellated textures are completely obliterated. This form of disease is also often described as exostosis; and no distinct boundary can, in many instances, be drawn between them. Were the term exostosis restricted to the exuberant growth of bone on the surface of bone, it would be easy to draw a line of distinction between it and chronic inflammation, which first causes an effusion of lymph; but when, according to Boyer, it is also made to comprehend the enlargement of a part or of the whole of a bone, it must be confessed that all chance of distinction is lost. Chronic inflammation just acts on bones as it does on soft parts, in the way of causing enlargement of their substance; neither do the former more than the latter ever regain their original size, after the undue action has ceased. Chronic inflammation of the periosteum sometimes occurs alone, and is liable to be mistaken for an affection of the bone; this membrane will even have ossific deposits along its inner surface, while the bone beneath remains perfectly free from disease.

Chronic inflammation, as it is seen in bones, admits of several modifications, arising from the state of the constitution as well as the nature of the local action. We have briefly alluded to two of these under the heads of enlargement of bones and venereal nodes, and another has been described under the name of *caries*. This word is derived from χειρω, signifying to abrade, and is best expressed in English by the word rottenness. No definition has been given which accurately describes this condition of a bone, yet the phrase caries has been in use for many centuries. It has been generally employed to signify ulceration of a bone, but there are several varieties of ulceration which ought not and cannot be comprehended under it. For example, there is a healthy kind of ulceration in bone, resulting from active inflammation which has ended in suppuration, where granulations spring up from the osseous structure, and the resources of the constitution are adequate to repair the breach. This state is not to be mistaken for caries. Again, a portion of dead bone is separated from the living by a process of ulceration or ulcerative absorption; but neither ought the term caries to be applied to this condition of the parts. We would define caries to be an unhealthy species of ulceration

occurring in the spongy or cancellated tissue of a bone, preceded by chronic inflammatory action, showing little or no disposition to heal, often arising from no evident cause, and for the most part taking place at an early period of life. To this definition it might be objected that caries is occasionally the consequence of acute inflammation, as in some cases where purulent matter has been formed in the ends of long bones; yet it may be replied that, in such, the disease first becomes chronic, and the granulations fall away before the state of caries succeeds.

Caries is confined to the spongy or cancellated osseous tissue, and is therefore generally met with in the round bones, as the vertebræ, those of the wrist or ankle, or the heads of the bones composing the extremities. It never occurs in the dense external wall, as the tables of the cranium or the shafts of the cylindrical bones, unless previously expanded and opened out by chronic inflammation; and even then, it has been considered as a sort of pseudo-caries, because it differs from the original in being more easily corrected. The fact of caries only occurring in the loose portion of a bone, is of importance in enabling us to separate it from necrosis or death of a bone, an affection with which it was long confounded. The distinction between these, in one point of view, is simple. In caries, the vital principle is still adherent; in necrosis, it is extinct. The two are, however, sometimes combined, as we observe in the heads of the long bones, and in some of those of the face when arising from syphilis. The absorption of a bone from pressure, as from an aneurismal tumour, or an abscess, has likewise been mistaken for caries; but, in such cases, there is simply a removal of the osseous particles without any ulcerative action, and as soon as the pressure ceases the absorption stops.

Whenever we have an opportunity of watching a case of genuine caries from the commencement, we invariably find that it is made up of two or three distinct stages, namely, one of chronic inflammation—another of secretion of pus—and a third of ulceration. The spongy substance of the bone is, in the first place, affected with inflammation; then purulent matter is effused; and this finally makes its way to the surface by causing ulcerative absorption of the external compact shell—the periosteum, and the integuments covering them. The early stage of caries is attended with considerable constitutional disorder; as soon as the confined pus is discharged, the irritation subsides; hectic fever subsequently ensues, under which and the secretion from the bone, unless the disease is removed by nature or art, the patient's strength is gradually undermined. We will treat more fully of caries when we come to the subject of ulceration.

Another modification of chronic inflammation in bone occurs in individuals of a scrofulous constitution. Here the disease originates in the articular extremities of the long bones, and in the spongy texture of the bones of the carpus and tarsus; and gives rise to such formidable disease of the joint as to have received the

name of the scrofulous white swelling. Like all other affections connected with scrofula, it is incidental to young subjects; sometimes commencing in different parts of the body at the same time, or in succession, and often complicated with enlarged mesenteric glands, tubercles in the lungs, and other signs of a cachectic diathesis. When the bones are examined in an early stage of the complaint, the cancelli are found filled with a transparent fluid, the earthy matter is partially removed, and there is increased vascularity. As the disease advances, the transparent fluid is removed, and a yellow cheesy-looking substance, resembling what is met with in the absorbent glands, is deposited in its place; so little phosphate of lime now remains behind, that the bone may be cut with a knife; and when macerated and dried, it is light and easily crushed. There is no enlargement or increase in the thickness of the diseased bone, the cells are merely filled with the peculiar scrofulous deposit, while the proper osseous particles are removed. When the inflammation reaches the articular surface of the bone, vessels carrying red blood are prolonged into the cartilage; and this structure, being incapable of resisting diseased action any length of time, is absorbed. The ulceration of the articular cartilage commences in distinct points on the side next the bone, and spreads through its substance till it reaches the synovial membrane reflected over it; thus the disease of the bone is communicated to the joint.

While these changes are going on internally, the tissue on the outside of the articulation becomes diseased. The cellular substance is thickened and loses its cohesion; a serous fluid and afterwards coagulating lymph are deposited between the capsule and the fascia which surrounds it; hence the swelling, at first soft and elastic, is succeeded by a firm colourless enlargement of the joint. The distension and subsequent induration of the soft parts have given rise to the opinion that the heads of the bones were enlarged; but when the other tissues are dissected away, such change is rarely if ever observed. As the disease advances abscesses form both within and without the articulation, making their way to the surface by circuitous routes, and discharging a thin pus, mixed with the coagulated flaky substance met with in scrofulous cases. When a probe is introduced through any of these openings, it is brought into contact with the diseased bone; and, frequently, portions of the latter are detached, creating great irritation before they are discharged. In some cases the purulent collections are confined to the cellular tissue, and have no communication with the cavity of the joint—the ulcerated openings healing up in one part and breaking out in another.

As long as the affection is confined to the head of the bone, there is only a deep-seated pain complained of, with stiffness on moving the joint; but, when the cartilages are involved and purulent matter is formed within the articulation, the local suffering is severe, and hectic fever, in its most aggravated form, sets in. The

progress of the disease varies extremely in different cases. In some, the morbid changes advance rapidly, abscesses appear quickly, and the articulating apparatus is destroyed within a few months from the commencement; in others, years will elapse after the joint has been affected, and yet no suppuration may ensue. The effusion of pus into the interior is a circumstance of great importance in all diseases involving a joint. Even under the most unfavourable circumstances, when the parts are kept in a state of absolute rest, the cavities of the abscesses may contract, the sinuses heal up, the ends of the denuded bones throw out granulations, and the patient escape with a stiff or anchylosed joint. The chance of a natural cure, however, is much less where the bones are affected by scrofulous inflammation, than where their articulating extremities are rendered carious by disease extending to them from the neighbouring structures; and this hope is still farther lessened—from the tendency the inflammation has to spread—where the joints of the wrist or ankle are affected, than where those of a less complicated description, as the knee or elbow, are the seat of disorder.

The popular phrase white swelling, therefore, has been indefinitely applied to several chronic affections of the joints; as where the disease has commenced in inflammation or disorganisation of the synovial membrane—in ulceration of the cartilages—or in scrofulous deposits in the loose osseous tissue. However much blended together these morbid changes may seem when a joint has been amputated and laid open, careful observation has proved that the affection usually begins in one particular tissue, and spreads by long continuance to the neighbouring and external parts. We hence find that the joint takes on different forms of swelling, and the case is attended with different symptoms, according as the one or the other structure is *primarily* affected; and that, of however little practical utility such knowledge in a late stage may be, yet we can tell with tolerable certainty at the commencement, what part of the articular apparatus is diseased, and how far there may be any hope of the morbid changes being arrested.

THE END.

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